Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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Vol. 128 **March 15, 1956** **No. 6**

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*Gray, C. W., Norden, Carl J., Jr.: Erysipelas Vaccine Avirulent (Eva)—A New Agent for Erysipelas Control. J.A.V.M.A., 127, (1955): 506-510.

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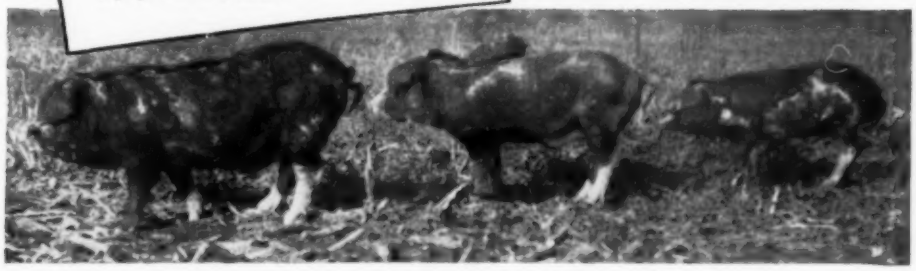

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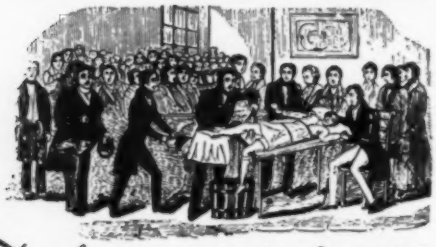

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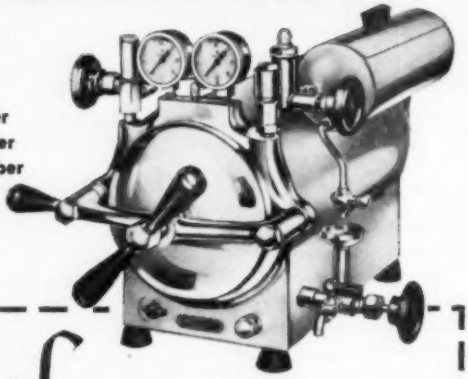

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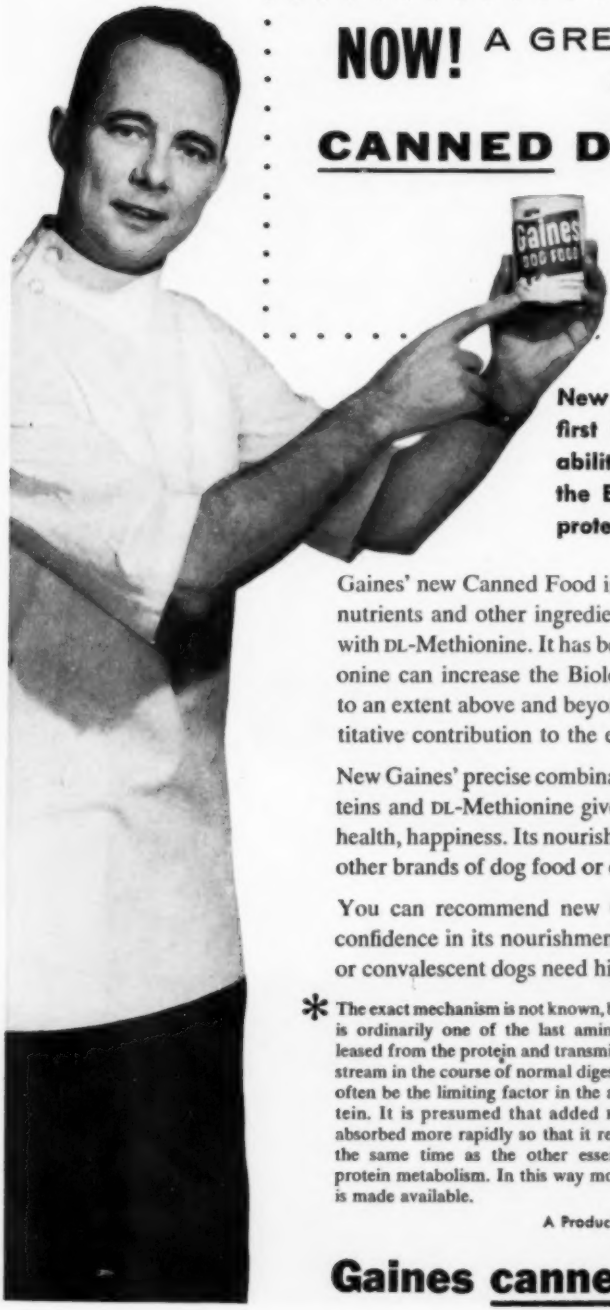

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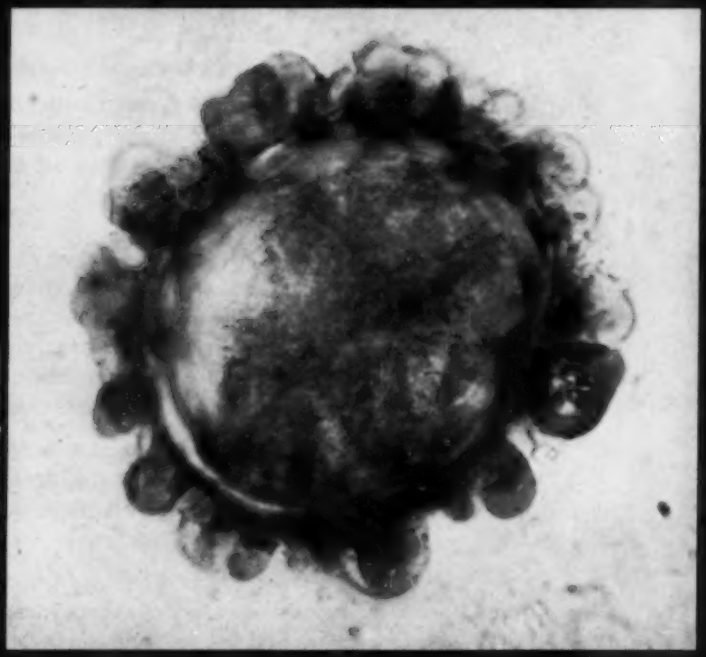

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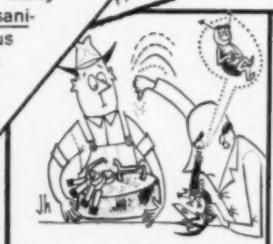

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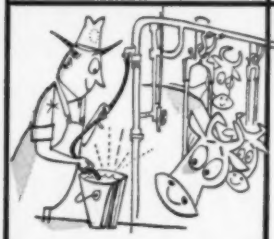

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News From Washington

H.R. 8320, as presently amended, provides an additional $2 million for year ending June 30, 1956, and $20 million for each fiscal year 1957 and 1958 to continue the accelerated brucellosis program. The AVMA was officially on record with the Committee on Agriculture and Forestry as requesting a substantial increase in funds for the eradication of brucellosis.

* * * *

Three identical bills, H. R. 8599 (Priest, D., Tenn.), H.R. 9006 (Hayworth, D., Mich.), and S.3176 (Murray, D., Mont., for himself and 7 other senators), would amend the Federal Food, Drug, and Cosmetic Act to establish a poultry inspection service as a division of the Food and Drug Administration, Department of Health, Education, and Welfare, and to require compulsory inspection. (Note. — Poultry Inspection, as well as grading, now on a voluntary basis, is presently conducted by the Agricultural Marketing Service, U.S.D.A.)

* * * *

ARS budget requests for fiscal 1957 are as follows: animal disease and pest control, $9,349,100 (excludes funds made available from the Commodity Credit Corporation for accelerated brucellosis program); meat inspection, $15,745,000; animal disease and parasite research, $3,159,160; foot-and-mouth disease research (Plum Island), $3,993,000.

* * * *

H.R. Concurrent Resolution 204 and S. Concurrent Resolution 69, introduced respectively by Congressman Priest (D., Tenn.) and Senator Hill (D., Ala.), would commemorate the 50th Anniversary of the Pure Food, Drug, and Cosmetic Law and the Federal Meat Inspection Act, respectively, and authorize and request the President to issue a proclamation designating the week beginning June 24, 1956, as National Pure Food, Drug, and Cosmetic and Meat Inspection Laws Week. Unfortunately, in House Concurrent Resolution 204, the words "meat inspection" were not included in the name of the week contained in the authority and request. AVMA, in testifying before the House Judiciary Committee, called attention to this error of omission, and requested the Resolution be amended.

* * * *

The AVMA representative testified on February 16 before the House Armed Services Committee, requesting inclusion of veterinarians in provisions of H.R. 8500. The bill, among other things, would give medical and dental officers four years of constructive service credit, and one year additional to physicians who had completed internship, and the service so credited would apply to grade and longevity pay. The bill would amend the Army-Navy-Public Health Service Medical Officers Procurement Act of 1947, the Career Compensation Act of 1949, and certain parts of the Officer Personnel Act of 1947. The subcommittee, in reporting the bill, according to reports, went further by recommending additional pay for medical and dental officers, scaled to years of service, and also the grade of lieutenant-general for the Surgeon General of the Army, the Navy, and the Air Force. (Note—House Armed Services Committee favorably reported H.R. 9428 as a substitute bill for H.R. 8500.) The subcommittee did not include veterinarians in any of the provisions, the reason being, so reported, that no procurement problem exists with respect to obtaining veterinarians for the Army or the Air Force.

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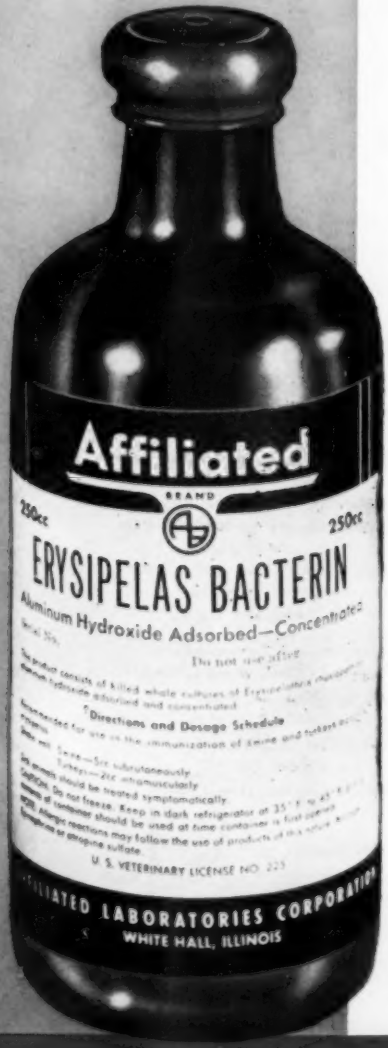

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JOURNAL
of the American Veterinary Medical Association
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Swine Erysipelas. I. A Discussion of Experimentally Induced Disease

DENNIS SIKES, D.V.M., Ph.D.; GEORGE M. NEHER, Ph.D.;
L. P. DOYLE, D.V.M., Ph.D.

Lafayette, Indiana

SWINE ERYSIPELAS is an infectious disease of hogs, sheep, goats, cattle, horses, turkeys, chickens, and man caused by the bacterium *Erysipelothrix rhusiopathiae*.[2,6,9,11,12] The disease in swine is known by other names such as red fever, and "diamond" skin disease, while in man it is sometimes called Rosenbach's erysipeloid. In swine, the disease is characterized by an acute septicemic course which later may become chronic if the animal survives, or it may pursue a chronic course from the onset, affecting the joints and heart. In man, infection of the skin, and later the heart and joints, may occur.

Swine erysipelas is widespread throughout Europe and has spread to all regions of the United States since 1921 when it was reported by Creech.[4]

The *Ery. rhusiopathiae* organism has the ability to survive in adverse environment outside the animal's body. Hettche[8] reported the organism was able to survive four to five days in drinking water and ten to 14 days in sewage. A saprophytic existence outside the animal's body has been suggested by Van Es,[16] Barber,[3] and Gledhill.[6] Thus, the hazards of potential spread by physical means in addition to animal carriers makes this disease especially difficult to control.

The experimental reproduction of erysipelas in swine with any degree of consistency has been limited. Kobe,[10] in reviewing Loeffler's original work done on swine erysipelas during the years 1882-1883, reported that he injected 13 hogs with tissues from swine erysipelas-infected mice and with cultures of *Ery. rhusiopathiae*. Only 1 of the 13 showed any significant symptoms and the postmortem findings suggested this pig died of some cause other than the injection of culture. Creech[4] used 22 pigs which were negative to the agglutination test for *Ery. rhusiopathiae* and artificially exposed them to cultures by feeding, intravenous, subcutaneous, and intraperitoneal inoculations. Five of the 22 pigs died and the organism was isolated from their spleens, livers, kidneys, and blood. Six pigs survived after showing symptoms of the disease while 11 showed no evidence of the disease. Schoening *et al.*[12] were able to reproduce acute and chronic erysipelas in 10 hogs with splenic emulsion and cultures taken from swine dead of erysipelas.

Sikes *et al.*[14] were able to reproduce swine erysipelas in 79 of 111 shoats 3½ to 4 months of age by scarification, subcutaneous, intramuscular, intravenous, intra-articular, and intradermal injections. Thirty-eight (34%) died of swine erysipelas, 41 (37%) developed advanced chronic arthritis, and 32 (29%) resisted exposure. Also, of 59 shoats which were placed in contact with these artificially exposed animals, 15 (25%) died of swine erysipelas, 8 (14%) developed chronic arthritis, and 36 (61%) resisted exposure. It was believed that the success in producing infection was due to three factors: (1) using animals of a most susceptible age (3½ to 4 mo.) from herds in which no clinical signs of swine erysipelas or arthritis had ever been observed; (2) by exposing the susceptible swine to smooth-type colonies of *Ery. rhusiopathiae* which (3) were grown at reduced oxygen tension in a semisolid medium enriched with 10 per cent horse serum.

The clinical manifestations of swine erysipelas are varied depending upon the degree of susceptibility of the animal and the pathological changes produced in the vital

From Purdue University, Lafayette, Ind. Dr. Sikes is now with the Department of Pathology and Parisitology, School of Veterinary Medicine, University of Georgia, Athens.

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Fig. 1—Extensive erythema of the skin of a white pig which died seven days after being exposed by contact to swine infected experimentally with Erysipelothrix rhusiopathiae.

tissues of the body, such as the blood, blood vessels, heart, kidneys, liver, lymphatic tissue, and adrenal glands. It is possible to recognize certain clinical forms due to the nature and course of the disease and to make a separate description of these forms. The disease may be acute or chronic with subdivisions of each form, depending upon the portion of the body most severely affected.

SYMPTOMS, PATHOLOGY, AND COURSE OF THE INDUCED DISEASE

In the acute septicemic form of the disease, the animal usually develops a fever of 106 to 108 F., soon becomes listless, and has a loss of appetite. If the fever remains high for 24 hours, the animal exhibits a rapid, shallow, panting type of respiration. Numerous large purple areas usually develop over the body and are most evident in nonpigmented skin (fig.1). In these animals, a marked leukopenia with a monocytosis is present. The animal is suffering from acute bacteremia and the organism can be isolated from the blood; if the fever subsides in a few hours, the animal may live; if, however, the temperature remains high for 36 to 48 hours, death is almost a certainty. If these animals are prostrate, as many are by 48 hours, they may drink water if held up or if a hose is placed in the mouth. These animals usually die in two to four days. At necropsy, the organism can be isolated from all tissues of the body including the brain and bone marrow. Extensive destruction of the small blood vessels has occurred, allowing blood to escape into the tissues. The loss of serum proteins and dehydration of the body apparently hasten death. In those that survive, there are usually symptoms suggesting muscle soreness and tender feet. Definite symptoms of arthritis are likely to develop later.

Fig. 2—A dry gangrene type of skin lesion of a pig 32 days after intra-articular exposure to Erysipelothrix rhusiopathiae.

In those which develop the urticarial or "diamond" skin form, the fever rarely exceeds 106.5 F., persists for one or two days, and promptly drops to about 104.0 F., but may remain slightly elevated for several days. The affected areas of the skin may be rhomboidial in outline or diffuse. The area is first a wheal with blanching of color which soon becomes erythematous, edematous, and raised above the surface of the nonaffected skin. These wheals may coalesce and form huge areas of degenerate skin and slough off as dry gangrene (fig. 2), or they may subside after 36 to 48 hours. In either case, chronic joint infection usually results and the animal becomes lame. The blood cell changes are not as pronounced in these animals as in the acute septicemic form. Agglutins usually appear in the blood in three to five days postexposure. If the disease remains chronic, a positive titer of 1:100 or more usually is present.

In swine with chronic arthritis, which seem particularly unthrifty (fig. 3), extensive vegetative endocarditis, involving the tricuspid and to a lesser extent the bicuspid valves, is sometimes observed at necropsy. The vegetation may be large enough to occlude the aperture (fig. 4). The authors regularly isolated *Ery. rhusiopathiae* from these lesions in experimentally infected animals. (Vegetative endocarditis occurs in swine under natural conditions from which this microorganism can not be isolated.)

The chronic arthritic animals are usually survivors of a previous acute infection, often the urticarial form of the disease. An

acute arthritis develops in two to three weeks postexposure, with effusions which distend the joint capsule. The animals are lame and constantly shift their weight from leg to leg while standing. The appetite is poor and a thermal response is usually not detectable *per rectum*. However, the swollen joints are hot, sensitive to touch, and painful. In two to three weeks, the acute swelling is reduced but the lameness persists and in many cases one limb may not be used to bear weight. If the infection persists for two months or longer, the chance of recovery is slight. At this stage a marked periostitis, osteitis or, in rare severe cases, osteomyelitis is present in the bones forming the affected joints. Marked thickening of the joint capsule (fig. 5 and 6) and osteophytic formations with ankylosis may develop. In many animals, the infection soon disappears following ankylosis, but the pathological process continues with periods of exacerbation and remission. The authors were unable to isolate the organism from such joints after 226 days and rarely after six to seven months postexposure. The periods of exacerbation and remission vary considerably and may be two weeks or more. An occasional spontaneous recovery occurs after several months, but the joints remain enlarged.

A comparison of the arthritis of swine due to *Ery. rhusiopathiae* infection and rheumatoid arthritis in man shows many similarities. The anatomical changes in the joints of both diseases show clear macroscopic and microscopic similarities according to Collins and Goldie.[4] The pathological changes in both diseases are essentially proliferative, nonsuppurative, and have a tendency toward granulomatous proliferations of the synovial membrane. Pannus formation and infiltration of the tissues with lymphocytes forming focal collections are observed in both diseases. Both show destruction of cartilage at the site of pannus attachment and subchondral cellular reaction. Intra-articular fibrous adhesions and rarefaction of the bone, revealed by radiograph, may occur in both diseases, but gross destruction of bone does not occur in either. Perhaps no further analogy between the two diseases should be drawn until the cause of rheumatoid arthritis in man is known. This arthritic condition experimentally produced in swine by the authors resembles the naturally occurring rheumatoid disease of swine as reported by Doyle[5] in 1951.

Fig. 3—The 7-month-old shoat on the right had been affected for three months with experimentally produced chronic erysipelas with polyarthritis. It weighed 152 lb.; its unexposed litter mate, 268 lb.

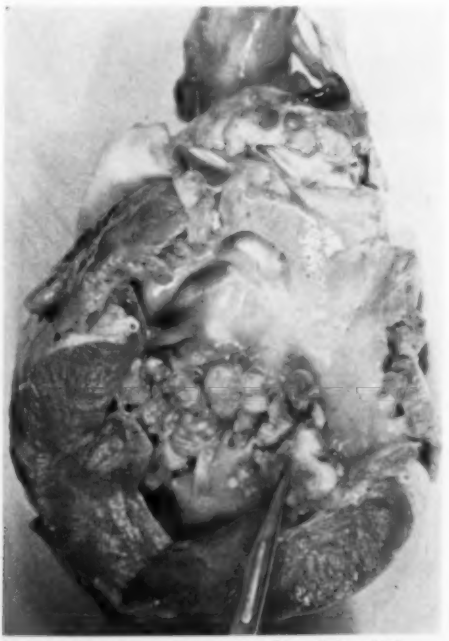

Fig. 4—Extensive vegetative endocarditis which resulted from intra-articular exposure of the pig to Erysipelothrix rhusiopathiae 112 days previously. The large vegetative masses which occluded the heart valve aperture probably caused death.

NOTES ON TREATMENT

The natural course of swine erysipelas in a herd varies, so the problems of control and therapy are not simple. The course of the disease, in a herd, may extend over several weeks or months with a few new cases appearing at regular intervals. Futhermore, since many affected animals recover spontaneously, evaluation of therapy may be difficult.

In the acute septicemic form of the disease, the use of anti-swine erysipelas serum administered concomitantly with penicillin is indicated if therapy is given at the onset of the elevation of temperature. This is important for two reasons: (1) to reduce the fever and to fortify the body defenses, since a marked leukopenia is prevalent in this stage, (2) and to protect the joints against localization of the organism in them. (The authors used this regimen of therapy in six severe outbreaks of the disease and less than 1% of the treated animals developed chronic arthritis.)

Fig. 6—A pair of front legs removed from a shoat which was exposed intravenously to Erysipelothrix rhusiopathiae 90 days prior to necropsy. Note the marked swellings of the carpal joints.

Fig. 5—Opened arthritic tarsal joint of a pig. Note the thickened joint capsule and the inflamed and hypertrophied synovial tissue.

Moving the nonaffected swine to a clean pasture is advisable to reduce the chance for further exposure. Whether or not this can be done, a dose of anti-swine erysipelas serum is indicated for the unaffected animals. Rarely does serum fail to protect the remaining animals for three to four weeks.

In cases of advanced chronic polyarthritis due to *Ery. rhusiopathiae* infections, cortisone has shown some promise in reducing the severity of the lameness, but does not seem to provide permanent relief. Only temporary clinical improvement resulted when Sikes *et al.*[15] treated 3 advanced cases of chronic polyarthritis due to *Ery. rhusiopathiae*. The respective dosage used was 75 mg. subcutaneously daily for four weeks, 50 mg. daily for one week, and 25 mg. daily for one week.

Adrenocorticotropin had some merit in the treatment of early cases of arthritis,

i.e., of three months' duration or less; however, the hormone was without appreciable benefit to swine with more advanced chronic arthritis.

SUMMARY

The literature pertaining to the experimental production of swine erysipelas is briefly reviewed and the symptoms, pathology, and clinical course of the disease is described. Acute and chronic erysipelas has been experimentally reproduced consistently in swine by six artificial routes of exposure and naturally by contact. In advanced chronic polyarthritis, the organism could not be isolated after 226 days and rarely after six to seven months postexposure. Many pathological changes of the joints in chronic polyarthritis due to *Erysipelothrix rhusiopathiae* infections resemble those of rheumatoid arthritis in man.

Anti-swine erysipelas serum and penicillin given at the onset of the acute phase of the disease proved to be effective in treating acute septicemic swine erysipelas. Although cortisone therapy will not eliminate chronic arthritis in swine, it did cause a remission of symptoms in the affected joints.

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Sixty Years of Erysipelas Control in Germany

The simultaneous serum and culture (*Erysipelothrix rhusiopathiae*) method of vaccination against swine erysipelas was introduced by Lorenz in 1893. Pasteur previously had produced a vaccine, supposedly weakened by animal passage, but "vaccination" erysipelas frequently followed its use. Other methods of immunization tried in various countries during this long period included: scarified skin inoculation; cultures weakened with agents such as saponin or trypoflavin; avirulent cultures; and killed vaccines.

Since 1947, a concentrated vaccine (*konzentratvakzin*) developed by Traub has been used extensively. It is produced by culturing certain strains of the organism, then killing them and adsorbing the immunizing substances to aluminum hydroxide.

The simultaneous vaccination, when first used, was frequently followed by vaccination erysipelas. The culture may have been too virulent, but it is now assumed that latent infection could have sensitized the animals so that vaccination with any culture would set up active infection.

The author (Schmidt), after many disappointments due to overvirulence or ineffectiveness, produced, in 1925, a satisfactory vaccination culture by combining two strains. Statistics compiled on its use, with serum, on about 200,000 swine annually for 16 years (1927 to 1942) showed a vaccination loss of only 0.003 per cent, the highest being 0.01 per cent in 1932.

The immunity record was also satisfactory, losses from erysipelas averaging 0.116 per cent until 1933, and only 0.037 per

cent after that. The dose for pigs under 50 kg. (100 lb.) was raised, in 1933, from 0.5 cc. so all pigs received 1.0 cc. of culture. These statistics, which refute contrary reports, are based on records of federal compensation paid for all laboratory-confirmed erysipelas losses in Mecklenberg since 1927.

The immunity period was estimated at five months after simultaneous vaccination alone, and at 12 months after a second (booster) inoculation (interval not given) with culture alone.

However, in 1954, while using the same standard and tested products, erysipelas, sometimes fatal, often followed the second inoculation. In some cases, the first vaccination had been with the concentrated vaccine so perhaps some outside factor, possibly the unusually warm weather, had interferred with the establishment of immunity. Of the swine prevaccinated by the simultaneous method, losses occurred only when the two agents were premixed, 1 part of culture to 4 or 5 parts of serum, and given as a single injection.

Failure could have been due to the resulting smaller dose of culture, the dose of the mixture being reduced to 3 cc. for small pigs, or due to its deterioration while being transported in the mixture in hot weather. More probably it was due to the effect of the immune serum on the organisms in the culture, possibly agglutination. In one area, two veterinarians were allowed to test this premix method but when an increase of "insufficient protection" became apparent, it was discontinued there.

CULTURE SEEMS HARMLESS

The merits of the simultaneous vaccination method and the concentrated vaccine (bacterin) method are compared. The latter has the advantage of being unable to spread infection. However, in view of the ubiquitary occurrence of *Ery. rhusiopathiae*, this seems insignificant. Moreover, during the 30 years, this culture has been given alone (illigitimately) without causing infection. Also, the strains used in vaccinating are probably "much more harmless" than the organisms "naturally deposited" in the throats and intestines of swine. And regardless of the method by which they had been vaccinated, it was found that most swine nevertheless became carriers during the summer months. Thus the theory that the standard vaccinating culture was dangerous because it spread infection would seem to be refuted.

Disadvantages of the concentrated vaccine (bacterin) are: (1) It gives no protection for about eight days; (2) serious suppurations (abscesses) frequently are produced by the aluminum hydroxide; and (3) this vaccine can be responsible for erysipelas developing in "allergic" or latently infected swine.

For an accurate evaluation of the two methods, the serum and culture must be injected at separate points and 1 cc. of culture must be given regardless of the size of the pig—*E. Schmidt, Monatsh. f. Vet.-med., Sept. 15, 1955.*

Treatment of Swine Erysipelas in Germany

Opinions among practicing veterinarians and authors, in Germany, differ as to the most effective treatment for swine erysipelas. This is "astonishing" since the disease long has been common; it caused a third of all swine losses for which insurance compensation was paid in the district of Leipzig in 1953.

To find the best treatment, it would be ideal to induce erysipelas, then test and compare various methods. However, the disease might not correspond with field cases and too many animals would be required. Therefore, a number of veterinarians were questioned about results with various treatments and their answers compiled.

Because of its variability, erysipelas is not easily classified. It, therefore, should be considered as a local infection with a general reaction and a tendency to develop into an acute, subacute, or chronic sepsis, with endocarditic and rheumatoid consequences in the latter. The "supposedly local" diamond skin lesions are associated with septicemia, and diseases of the joints and endocardium result when resistance curbs the septicemia. Because of this "multicolored clinical picture," comparisons of therapy to be valid must include large numbers of affected animals.

The results of the survey follow.

Serum.—Recoveries were more frequent with immune serum produced from swine (40%) than from horses (27%), but the difference was not significant. Large doses produced some side-effects. Intravenous ad-

ministration resulted in little benefit and more losses from shock.

Penicillin.—Recoveries increased significantly (to 90%) when penicillin was added to the serum. The number of animals in which the temperature was normal in 24 hours after treatment also increased (31 to 80%) and consequently the number with chronic postrecovery lesions was reduced (28 to 5%). Relapses are reported following therapy with penicillin alone. Therefore, to prolong the effect, slowly absorbed "depot" penicillin should be given with the quick-acting "water soluble" penicillin. However, in experimental cases, unlike other diseases, a prolonged serum level of penicillin seemed unnecessary.

Other therapy.—Nonspecific drugs tested (omnadin, sulfonamides, cardiac stimulants, and laxatives) seemed to be superfluous.

Conclusion.—The use of penicillin is "absolutely imperative."—*L. Felix Muller, Monatsh. f. Vet.-med., Oct., 1955.*

Swine Erysipelas. II. Prophylactic Effect of a Commercially Prepared Bacterin in Swine

DENNIS SIKES, D.V.M., Ph.D., GEORGE M. NEHER, Ph.D.;
L. P. DOYLE, D.V.M., Ph.D.

Lafayette, Indiana

PASTEUR and others used a bacterin in attempts to protect swine against *Erysipelothrix rhusiopathiae* infections. From time to time many other bacterins and vaccines have appeared which were supposed to have good antigenic properities, and to protect swine against *Ery. rhusiopathiae* infections but all have been found to have limitations for one reason or another. Perhaps this was because the mouse and pigeon have been used to evaluate these biological products since the disease could not be experimentally reproduced with consistent regularity in swine, the natural host. Due to the consistency with which acute swine erysipelas has been reproduced, as reported by Sikes *et al.*,[1] it was thought desirable to use swine to evaluate a commercially prepared bacterin.

MATERIALS

A total of 41 shoats, 4 months of age and weighing 115 to 125 lb. each, was used. They came from a purebred Chester White herd in which no clinical signs of swine erysipelas or arthritis have been observed since it was established in 1941. Two plans of management were used. In plan A, the animals were kept in a barn in pens with cement floors. Self-feeders or feed troughs were used. In plan C, the animals were placed on a two-acre alfalfa and Ladino clover pasture with hog houses, shade, and self-feeders. Hereafter reference will be made to management plan A or C.

The feed consisted of a well-balanced ration containing adequate minerals, vitamins, fat, and protein. When feed throughs were used, the hogs were fed liberally twice daily.

Tryptose agar (Difco) was used for primary isolation of cultures. Later, stock cultures were incubated for 24 hours in a semisolid medium (Difco, brain, liver, heart—pH 7.2) plus 10 per cent horse serum and then placed in a refrigerator. Transfers were made at monthly intervals.

METHODS AND RESULTS

Experiment 1—Protection of Swine at Six-Weeks Postvaccination.—Using plan A, each of 6 shoats was injected subcutaneously with 5 cc. of the bacterin in the axillary region. Four other shoats were placed in contact with them. Six weeks later, each animal was challenged with 10 cc. of a 24-hour culture of the organism intravenously in the anterior vena cava. Two weeks later, when the acute phase of infection had subsided, the animals were turned on the alfalfa and Ladino clover pasture. Two months after challenge, when the infection had become chronic and the animals were suffering from chronic polyarthritis, 6 more normal shoats were added to the group to

From Purdue University, Lafayette, Ind. Dr. Sikes is now with the Department of Pathology and Parasitology, School of Veterinary Medicine, University of Georgia, Athens.

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see if they too would develop erysipelas and arthritis.

All six of the vaccinated shoats showed a mild febrile response on days 1 and 2, postchallenge. One of the 6 shoats died on postexposure day 79 with vegetative endocarditis and chronic arthritis, and *Ery. rhusiopathiae* was isolated from the heart and carpal and tarsal joints. An extensive synovitis, synovial villi proliferation, and increased synovial fluid were present. The other 5 shoats developed chronic polyarthritis which persisted until the end of the 90-day experiment.

Three of the 4 contact shoats exposed with 10 cc. each of a 24-hour culture died of acute swine erysipelas on days 3 and 4, postexposure; *Ery. rhusiopathiae* was isolated from their kidneys, livers, and spleens. The fourth animal developed chronic arthritis which persisted for 90 days.

All of the 6 control shoats, which had been placed in contact with those with chronic arthritis, remained healthy for the 30 days until the end of the experiment.

Experiment 2—Protection of Swine at 12 Weeks Postvaccination.— Under plan A, each of 15 shoats was injected subcutaneously with 5 cc. of bacterin in the axillary region and 4 control shoats were placed in contact with them. Twelve weeks later, 8 of the vaccinated and 2 of the contact shoats were challenged with 10 cc. of a 24-hour culture of the erysipelas organism in the anterior vena cava. Six normal control shoats were placed in contact with these animals two months after exposure, as in experiment 1.

Four of the 8 vaccinated shoats, when challenged 12 weeks later, died with acute swine erysipelas by the fourth day. Necropsy revealed gross pathological changes typical of acute swine erysipelas and *Ery. rhusiopathiae* was isolated from the liver, kidneys, and spleen of each. The 4 remaining shoats had a high febrile response of 106.5 to 107.5 F. for two days and then the temperature gradually returned to normal. Many skin lesions appeared on the body during the acute phase of the infection. These lesions were erythematous areas of varying size, some of which were raised. Later, each animal developed acute polyarthritis which became chronic after three weeks.

Of the 2 shoats in contact with the bacterin-vaccinated group, 1 died of acute swine erysipelas on the fourth day after intravenous exposure, the other 1 became acutely ill but survived. It developed acute polyarthritis which later became chronic. The other 2 contact shoats, which were exposed naturally, became acutely ill, had many erythematous lesions on their bodies, and had temperatures of 106.0 to 107.0 F. for two days. They developed acute polyarthritis which later became chronic.

Three of the 7 bacterin-vaccinated shoats, which were exposed only by contact, developed acute swine erysipelas and had temperatures of 106.5 to 107.0 F. for two days. Many erythematous areas developed over their bodies, followed by acute polyarthritis which later became chronic. One of the 6 normal animals which had been placed in contact with these pigs with chronic polyarthritis developed acute swine erysipelas, similar to that described, and later developed acute polyarthritis which became chronic.

DISCUSSION

The inability to consistently reproduce typical swine erysipelas in hogs has been one of the most intriguing problems confronting investigators since the disease was first recognized. The practicing veterinarian recognized the disease, was able to treat the affected animals, and to aid in the control of the disease, but the research workers were not able to duplicate the typical naturally occurring disease in swine. As a result, the various biological products used as prophylactic agents could not be effectively evaluated. Even the system of giving crystal-violet hog cholera vaccine to cholera-susceptible shoats and then challenging them with live virus three weeks later to make them highly susceptible to *Ery. rhusiopathiae* infections, as practiced by Doyle in England, met with only partial success when employed by Thompson and Gledhill.[2]

Vallie[3] used a living, attenuated vaccine in 9 shoats without any deleterious effect. Twenty-four days later, they resisted challenge with a virulent culture of *Ery. rhusiopathiae* while all the control pigs developed high temperatures, but none died. No animals were exposed by contact.

This experiment suggests that some protection resulted from the vaccination; however, the virulence of challenge organism could be questioned since none of the control pigs died. In all of Vallie's experiments, the organism used for challenging had been grown on solid mediums in ordinary atmosphere until broth was inoculated 24 hours before the time of exposure. This technique disregarded the microaerophilic properties of *Ery. rhusiopathiae*.

In experiment 1, 3 of the 4 shoats used as contact controls died by the fourth day after intravenous exposure. The fourth one developed acute swine erysipelas followed by chronic polyarthritis. In contrast, the 6 bacterin-vaccinated shoats had a mild febrile response but survived and later developed chronic polyarthritis. These results indicate that, six weeks after vaccination, some immunity was present but not enough to give complete protection. This would seem logical since most antigens require approximately three weeks to produce the maximum antibody titer. A diminution of titer then occurs unless the animal is re-exposed to the antigen.

In experiment 2, 4 of the 8 vaccinated animals, challenged 12 weeks after receiving the bacterin, died of acute swine erysipelas.

The other 4 animals developed acute swine erysipelas and, later, chronic polyarthritis. Apparently, little or no immunity was present in these animals when challenged. In this experiment, the incidence of infection was 100 per cent and the death rate was the maximum obtained in all experiments at the dosage used (Sikes *et al.*[1]). One of the 2 susceptible shoats placed in contact with the vaccinated animals died of acute swine erysipelas after intravenous challenge. The other 1 developed acute swine erysipelas, followed by chronic polyarthritis. There apparently was no difference in susceptibility in the 8 vaccinated and the 2 unvaccinated contact animals when they were challenged intravenously.

Three of the 7 remaining vaccinated then naturally exposed animals developed acute swine erysipelas and later a chronic polyarthritis. This represents a 43 per cent infection rate. (This is higher than that observed in most natural outbreaks of the disease.) From the result of this experiment, it was concluded that no effective immunity was present 12 weeks after vaccination. The 4 animals which withstood the natural exposure probably were naturally resistant, as many swine seem to be.[1] (Field observations also support this belief.)

The 2 contact, nonvaccinated control animals developed acute swine erysipelas through natural exposure. Later they developed chronic polyarthritis. These results indicate that the environment had no effect on the development of immunity and that the bacterin was safe to use in a mixed group of hogs.

In experiments 1 and 2, a total of 12 shoats was placed in contact with the chronic arthritis animals two months after exposure. One of the 12 developed acute swine erysipelas and cronic polyarthritis later. These results indicate that susceptible swine should be moved from contaminated surroundings and from known affected animals. (Veterinarians have recommended this practice for many years with good results.)

It would seem that the bacterin gave some immunity for six weeks after vaccination, but that the protection was lost prior to the 12-week challenge. Van Es and McGrath[4] believed that anti-swine erysipelas serum would protect swine for two to three weeks, and that a more enduring immunity could be established by the simultaneous injection of a highly virulent culture of the organisms with the serum. This procedure has the disadvantage of producing active infection which may persist in swine, and they in turn may become active spreaders of the organism. This practice may have merit where the disease is enzootic. Many believe that such areas exist in central Europe and to a lesser extent in the United States. Many states object to the use of such a practice on the basis that the incidence of infection is not great enough. Until such natural enzootic infection occurs regularly, the wisdom of such a practice is doubted. The judicious use of anti-swine erysipelas serum, penicillin, and the moving of nonaffected animals to uncontaminated surroundings should control the disease.

Summary and Conclusions

A commercially prepared swine erysipelas bacterin was used to vaccinate 21

shoats 4 months of age. Six of these shoats, when challenged at six weeks after vaccination, had a febrile response and later developed chronic polyarthritis. Three of the 4 contact shoats similarly exposed died of acute swine erysipelas and the fourth one developed chronic arthritis. Of the 15 remaining vaccinated shoats, 8 were challenged 12 weeks later and 4 died of acute swine erysipelas while the remaining 4 developed chronic arthritis. One of the 2 contact shoats, similarly exposed intravenously, died of acute swine erysipelas, while the other 2 contact shoats, naturally exposed, developed chronic arthritis. Three of the 7 remaining vaccinated shoats, exposed only by contact, developed acute swine erysipelas followed by chronic arthritis, while the other 4 resisted exposure. Of the 12 normal shoats placed in contact with these animals with chronic arthritis two months postexposure, 1 developed chronic arthritis.

It is concluded that:

1) The bacterin was safe to use in herds where only a portion of the group was vaccinated.

2) Some protection existed six weeks postvaccination but it was not complete since all animals developed chronic polyarthritis.

3) There was no evidence of protection when the swine were challenged 12 weeks postvaccination.

Since 1 of 12 shoats became infected when housed in contact with swine which had been arthritic for two months, separation of nonaffected animals is indicated as a control procedure.

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Milk production per cow in Russia annually is about half that in the United States.—*Sci. News Letter, Jan. 14, 1956.*

This Is Part 1

This issue (March 15) of the JOURNAL is published in two parts. This is Part 1, as noted on the cover. Part 2, containing the Business Proceedings of the Ninety-Second Annual Meeting of the Association, held in Minneapolis last August, and the proceedings of the Preconvention Conference, is being mailed only to members of the Association.

Proprietary Drugs in Norway

As a safeguard to the public, only about 1,300 proprietary synthetic drugs have been registered in Norway compared with 25,000 such drugs in some countries. Even so, in several instances there is some overlapping due to the registration of several names for the same drug. Over 20 penicillin preparations are registered, but these are being reduced. Advertisement control and certain price controls are effected through registration.—*J.Am.M.A., Oct. 29, 1955.*

Since tranquilizing drugs, such as reserpine, have been in use at the Mississippi State Hospital, shock treatments for mental patients (11,320 in 1953-1954) have been discontinued.—*Sci. News Letter, Dec. 10, 1955.*

Veterinarian Contracts Anthrax.—Cutaneous anthrax developed on the left forearm of a Louisiana veterinarian five days after he had performed a necropsy on a cow dead of the disease. *Bacillus anthracis* was isolated from the lesion on the arm and from the cow.—*U.S.P.H.S. Release, Dec. 8, 1955.*

Spotted Alfalfa Aphid, A New Pest

A new alfalfa pest, an aphid about the size of a pinhead which hails from India and the Middle East and may have been brought in by airplane, was found in California in 1954. By late 1955, it had spread well through Missouri and Nebraska, killing 40 per cent of the alfalfa in Oklahoma. Because of rapid reproduction and migration, insecticides checked it only temporarily. The effect of its natural enemies, tiny wasps and a fungus, are being studied in California.—*Farm J., Jan., 1956.*

Surgery and Obstetrics
and Problems of Breeding

Succinylcholine Iodide as a Muscular Relaxant in Veterinary Surgery

C.-H. HANSSON, V.S.
Stockholm, Sweden

THERE HAS long been a need in large animal surgery for an anesthetic that is relatively risk-free but which, nevertheless, produces adequate muscular relaxation. No such preparation seems to exist at present. However, the recent discovery[3,4] of good muscular relaxants has provided a new approach to the problem of anesthesia of large animals. In man, d-tubocurarine has been used extensively both in surgery and in electroshock therapy.[13] In veterinary medicine, it has not been employed extensively, because of its toxicity and its cost. It has, however, found a limited application in equine surgery[2,17] and in canine surgery, particularly in intrathoracic operations.[6,10,14,16]

Consequently, when succinylcholine iodide was purified in 1951[12] and later marketed as celocurin,* experiments were started at our clinic to ascertain its usefulness in veterinary medicine.[8] The absence of toxic reactions[5,18] and its low cost make celocurin eminently suitable for veterinary use, even in large animals.[1,9] The pharmacological effect of succinylcholine iodide consists of blocking the neuromuscular transmission by depolarization of the muscle end-plates. Succinylcholine is broken down by cholinesterases in the body into choline and succinic acid. The duration of its effect is, therefore, related to a certain extent to the cholinesterase activity, which varies appreciably in different species of animals.[11]

CELOCURIN IN EQUINE SURGERY

Restraint by casting has always been a problem in equine surgery. Complications may arise, particularly in nervous animals. Moreover, it has often been difficult to obtain sufficient assistance. A series of experiments was therefore made to ascertain whether celocurin could be used to replace the classical method of casting.

For humane reasons, celocurin should not be used alone, but always with a sedative, such as a 6 per cent solution of nembutal.® To determine whether the procedure had any mental effect on the patients, a medium-sized horse was cast ten times, using celocurin after premedication with 18 ml. of 6 per cent nembutal solution injected intravenously. The injection of the sedative was then repeated and the horse was cast another ten times. The horse exhibited no signs of fear or apprehension, despite the fact that he was cast each time in the same manner. When the experiment was repeated without a sedative, the horse showed marked signs of apprehension, and after being cast three or four times, it was practically impossible to give him an injection.

Technique for Casting.—If the horse is to be laid on the left side, the head is lifted up and turned slightly to the right. A rope is put over the neck from the left side, then under the right foreleg, and up over the back to the left side. When the horse starts to stagger from the effect of celocurin, this rope is pulled gently. When the horse is falling, its head should be supported in order to avoid injury to the teeth and lips. If the hobbles have not already been put on, this is done as soon as the horse has fallen, and fettering of the horse is then completed.

Results.—In all experiments, when the dose of celocurin exceeded 0.15 mg. per kilogram of body weight, the behavior of the horses was practically identical in the initial stage. About 30 to 40 seconds after the injection, the head dropped and the third eyelid became visible. At the same time, the horse started to tremble and, after about another 30 seconds, it fell. With a dose not exceeding 0.18 mg./kg. of body weight, there was a superficial costo-abdominal respiration for about one minute, with respiration becoming relatively normal after about two minutes. With a dose of 0.2 to 0.25 mg./kg. of body weight, the respiration became superficial, was sometimes purely abdominal, and was usually associated with slight cyanosis and an increased pulse rate. The respiration rate

From the Department of Surgery, Royal Veterinary College, Stockholm, Sweden.

*Manufactured by Vitrum, Stockholm, Sweden.

TABLE 1—The Effect of Intravenous Administration of Celocurin in the Horse

Dose per kg. body weight (mg.)	Wt. of animal (kg.)	Total quantity of celocurin (mg.)	Quantity of nembutal solution (ml.)	Abolition of righting reflex	Duration[1] of effect (min.)	Respiratory arrest (sec.)	Cyanosis
0.13	790	100	30	+	5	—	+
0.13	770	100	30	+	5	20	++
0.15	650	100	30	+	5	20	++
0.16	520	85	18	+	4	—	0
0.16	490	75	20	+	5	—	0
0.18[2]	550	100	20	—	—	—	0
0.18	550	100	20	+	6	—	0
0.21	470	100	16	+	4	—	+
0.23	550	150	20	+	6	50	+++
0.27	550	150	20	+	9	90	+++
0.28[2]	530	150	20	+	8	90	0
0.30[2]	500	150	20	+	8	95	0

[1]The duration denotes the time during which the horse does not react with defense movements to pricks in the coronary region. This patient was a trained race horse. [2]Artificial respiration was given for one minute.

returned to normal after two to three minutes.

For draft horses and heavy horses, a dose of 0.13 mg./kg. of body weight was sufficient for casting (table 1).

Complications.—The only complication of importance was the respiratory arrest resulting from overdosage. However, with a reasonable dose, it never lasted for more than a minute. Although this respiratory arrest was not serious, it presumably could cause some discomfort. For experimental purpose, 0.3 mg./kg. of body weight of celocurin was given without artificial respiration. The horse exhibited moderate cyanosis only, and after about one and a half minutes spontaneous respiration started. Artificial respiration, which should be given to prevent any suffering, is performed most simply and effectively by rhythmic compression of the abdomen.

Celocurin has now been used routinely to cast about 500 horses in the past 18 months without any complications.

CELOCURIN IN CANINE SURGERY

The cholinesterase activity in the dog shows a wide range of variations, depending on the breed.[7] This factor was not considered here, the dosage being calculated according to the body weight irrespective of the breed. Nor were any determinations of the cholinesterase activity in the experimental animals made.

The clinical symptoms and dosage were recorded on the basis of experience with the first group in which the dogs were anesthetised with thiopental sodium injected intravenously. They were then intubated and anesthesia was continued with trichloroethylene.[14] The femoral nerve was dissected free and was connected to a stimulating electrode. The tibia was fixed by means of a clamp to a table stand; the tendon of the gastrocnemius muscle was then cut and the central end was connected by a thread to a spring-recording pencil. The blood pressure was measured in the femoral artery. Each experiment comprised at least 3 dogs of varying size and breed. Repeated injections of celocurin were given to each dog but at sufficiently long intervals to allow breaking down of the previous dose. Each experiment lasted for at least two hours.

TABLE 2—Survey of the Effect of Celocurin on the Neuromuscular Transmission, Ventilation, and Blood Pressure on Intravenous Injection in the Dog

Experiment (No.)	Celocurin (mg./kg.)	Max. degree of relaxation[1] (%)	Duration of effect (min.)	Ventilation[2] (%)	Duration of respiratory arrest	Variation in blood pressure (mm. Hg.)
I	0.022	25	2-3	100	0	−1
II	0.045	50	5-7	75	0	+4
III	0.060	50	6-11.5	50	0	+8
IV	0.066	>50	11-14	50	0	+15
V	0.085	>50	8-11	0	45 sec.	−8
VI	0.11	>50	18-21	0	10 min.	−6
VII	0.22	>50	23-27	0	10 min.	−9

[1]The degree of relaxation denotes the ratio of the amplitude of the normal muscle to that of the "curarized" muscle (see fig. 1). A degree of relaxation of 50 per cent usually suffices for clinical purpose.
[2]Ventilation denotes the difference between the expiratory and the inspiratory pressure in the trachea as a percentage of the basic value of the difference.

In the results as shown (table 2), each value is the mean of the values obtained in each experimental group.

A series of experiments was also made to study the effect of administration of celocurin by intravenous drip. Ten minutes before starting the experiment, an intramuscular injection of 2 per cent morphine solution with scopolamine was given, the dose being about 0.05 ml./kg. of body weight of a 0.04 per cent solution. The concentration of the celocurin solution was such that 40 drops per minute corresponded to 1.2 mg. The dose of celocurin was about 0.015 mg./kg. of body weight per minute. The blood pressure was measured.[15] If the administration was adjusted in such a way that satisfactory spontaneous respiration was maintained, the blood pressure never deviated from the basic value by more than 20 per cent.

If prolonged effect is desired and continuous intravenous administration is not feasible, repeated intravenous injections can be given at short intervals.

Prolonged administration of celocurin gives rise to marked hypersecretion which can, however, be completely eliminated by the intramuscular injection of 0.03 mg./kg. of body weight of a 1 per cent atropine solution.

To show the possibility of producing muscular relaxation in wild animals or in animals whose temperament makes it impossible to use the intravenous route, tests were also made on a number of dogs with the intramuscular administration of celocurin. The dosage was 0.12 mg./kg. of body weight. After about five minutes, the dogs were unable to walk, but respiration was still normal, and after a further two minutes forced abdominal respiration appeared, but without signs of cyanosis. About three minutes later, respiration was once more fully normal and after about another 30 minutes the dogs were able to run about completely unaffected. During these experiments, salivation was heavy.

Clinical Experience.—Celocurin has proved to be a valuable adiuvant to thiopental-trichloroethylene anesthesia. In this form of anesthesia, narcosis is often too superficial to produce the necessary muscular relaxation. Furthermore, trichloroethylene often causes a disturbing tachypnea. In such cases, there are two alternatives: (1) More thiopental sodium can be injected;

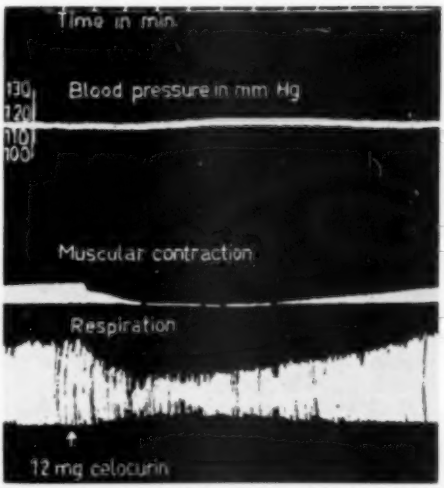

Fig. 1—The effect of intravenous injection (0.067 mg./kg.) of succinylcholine iodide(celocurin), in a dog weighing 18 kg., on the blood pressure, the respiration (recorded with a piston recorder connected to a tracheal tube), and the twitch of the gastrocnemius muscle excited by supramaximal shocks to the sciatic nerve every second.

or (2) a sufficient quantity of celocurin can be given to produce muscular relaxation and, in case of tachypnea, arrest of spontaneous respiration. The latter is definitely the method of choice, since a dog whose general condition is already poor is often unfavorably affected by reinjection of an anesthetic. It is thus possible, with the aid of celocurin, to keep anesthesia on a superficial level and still obtain satisfactory muscular relaxation.

Combined thiopental, trichoroethylene, and celocurin anesthesia has the additional practical advantage of more rapid recovery from narcosis than with nembutal and thiopental sodium.

Celocurin has been particularly valuable in intrathoracic interventions. This is because the paralysis of the diaphragm es-

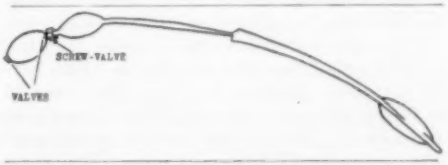

Fig. 2—Probe with pneumatic rubber bulb for occlusion of the esophagus of the cow.

sential for these operations can easily be produced without deepening the level of anesthesia. The usual dose is about 0.15 mg./kg. of body weight.

CELOCURIN IN BOVINE SURGERY

Preparatory to the use of celocurin in cattle, comparative investigations were made of the breaking down of celocurin *in vitro* in bovine and equine blood. The dose was then calculated for cattle and *in vivo* experiments were carried out. It was found that the calculated dose was far too high, and that lengthy apnea occurred. This could be controlled only by reducing the dose to about ⅛ of that necessary in the horse.

With a dose of approximately 0.02 mg./kg. of body weight, all the experimental animals behaved similarly: About 2 to 3 minutes after the injection, sleepiness and fibrillar muscle twitching appeared simultaneously. The legs then "folded up" and the cow fell, always slowly with the animal never hitting its head on the ground. In most cases, there was some residual tonus in the neck muscles, despite complete paralysis of the extremities.

The only factor that varied, and was extremely difficult to evaluate in cattle, was the duration of effect. As a rule, the animals lay for about 15 minutes before being able to rise, but often, although the power of movement seemed to have returned, the cow was disinclined to attempt to stand. The duration of the effect was considered as the time during which the cow lay, making no attempt to rise without urging.

With a dose of 0.025 mg./kg. of body weight and above, it is always necessary to anticipate apnea of varying duration. This is in no way serious if artificial respiration is given. However, this can not be done in cattle without simultaneously blocking the esophagus, as compression of the chest or abdomen forces, the fluid content of the rumen up into the esophagus and pharynx and the risk of aspiration is, thus, great. Occlusion of the esophagus is most easily done by passing a rubber tube provided with a balloon (fig. 2) through the nasal cavity into the esophagus. The balloon is then inflated to a suitable size. If it is too strongly inflated, reflex peristaltic movements are elicited in the esophagus, with concurrent hypersecretion. The tube should be attached to the halter, as it may otherwise be swallowed. Artificial respiration is then performed by rhythmic compression of the thorax.

As in the horse, celocurin should not be used in cattle without concurrent administration of a sedative. In experiments, as well as in clinical practice, 6 per cent nembutal solution has been used, the dose being about 15 ml. for medium sized cows and 20 ml. for larger animals (over 600 kg). This dose seems to be fully adequate to prevent any unpleasant sensation due to the muscle-paralyzing effect of celocurin.

Necropsies were performed on 30 experimental cattle after they had been cast with celocurin and slaughtered. Particular interest was then focused on the trachea and lungs. In no case in which the dose was below 0.03 mg./kg. of body weight, or in which occlusion of the esophagus was performed, were there any signs of aspiration of the ruminal content.

In clinical practice, the dosage can be fixed with sufficient accuracy after estimation of the animal's weight. A dose of 8 to 10 mg. is suitable for small-sized cattle (400-450 kg.). For medium-sized cattle (450-550 kg.) 10 to 12 mg. are required, and for large-sized cattle (550-650 kg.) 12 to 14 mg. If the dose is too small, ten minutes are allowed to elapse, after which the same dose of celocurin, increased by 2 mg. and dissolved in saline solution, is injected (the nembutal effect persists after the first injection). If, however, the dose is only slightly too small, putting the animal off balance often suffices to make it fall. After the cow has fallen, it is fettered and the operation is then performed under general or local anesthesia, according to the circumstances. Minor operations can be performed without fettering the cow.

SUMMARY

1) The dose of succinylcholine iodide (celocurin) necessary to cast a horse was found to be 0.18 mg./kg. of body weight in thoroughbred horses and 0.13 mg./kg. in draft horses. About 500 horses were cast with celocurin at the surgical department of the Royal Veterinary College.

2) The minimum dose in dogs was found to be 0.067 mg./kg. of body weight. In the dog, celocurin was found to combine well with thiopental-trichloroethylene narcosis.

3) In order to cast cattle, 0.02 mg. of celocurin per kilogram of body weight was found to be necessary. Only 50 cattle were

used and further clinical trials are needed. If a higher dose is injected into cattle, an esophageal probe must be employed to avoid aspiration of food.

4) Celocurin should be used only in combination with a sedative agent or with narcosis.

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Chronic bloaters are best treated by making a permanent rumen fistula. Under local anesthesia, remove an elliptical piece of skin and underlying tissues, about 3 inches long, high in the left flank, allow the rumen to bulge through, suture it to the skin, then remove a similar section of the rumen wall.—*V. J. Humphreys, D.V.M., Wyoming.*

Epidural Anesthesia in the Dog

Excellent caudal anesthesia resulted from the use of a 5 per cent procaine solution with adrenaline in 17 dogs and with an 8 per cent solution without adrenaline in 6 dogs, the dose being 1 ml. per 5 to 6 kg. of body weight. Analgesia and muscle relaxation posterior to the umbilical region developed in three to five minutes and lasted 90 to 120 minutes and without side effects. —*Vet. Bull., Oct., 1955.*

Parturition in the Cat and Bitch

By hysterectomy or necropsy of 16 cats with abnormal parturition, and of 23 experimental normal cats, the fetuses were found to be unevenly distributed in the horns. The first one expelled was from the horn with the greater number of fetuses and the first 3 came alternately from the 2 horns. What determined which came first when the fetuses were equally distributed was not clear. Similar observations were made on 39 bitches.—*Vet. Bull., Dec., 1955.*

Roosters Fertile After Stilbestrol.—Diethylstilbestrol implants (15 mg.) in 11-week-old cockerels did not affect their fertility at maturity. Those given the pill form of implants required more time to recover fertility than did those given a paste form. —*Poult. Sci., July, 1955.*

Pelvic Changes in the Pregnant Ewe

Dissection and radiography of 70 pregnant ewes in New Zealand revealed no relaxation of the pubic symphysis at any stage of pregnancy; relaxation of sacro-iliac joints and elongation of the sacrosciatic ligaments after two months of gestation; an increased flexibility of the sacrolumbar joint; and an increase both in the length and circumference of the vagina as parturition was approached.—*Vet. Bull., Oct., 1955.*

Clinical Data

Clinical Uses of Chlorpromazine in Veterinary Medicine

EMILIO ESTRADA, D.V.M.

West Haven, Connecticut

CHLORPROMAZINE (10-(3-dimethylaminopropyl) - 2 - chlorphenothiazine hydrochloride) (thorazine)* is a comparatively new drug which has been available to the medical profession in the United States for a little over two years. Its wide application in human medicine is attributed in large part to three main pharmacological properties: (1) antiemetic action; (2) central depressant action; and (3) enhancing effect on the activity of other drugs such as analgesics, anesthetics, and sedatives.

To date, there are few references to the clinical application of chlorpromazine in veterinary medicine. It would seem that this drug has application in small animal medicine and this study attempts to obtain data on: (1) the effect when employed as a preanesthetic sedative in conjunction with pentobarbital anesthesia; (2) the effect on nervous, unfriendly, or vicious dogs; and (3) its usefulness as an antiemetic. The observations reported here are based on the use of the drug on 157 dogs.

MATERIAL AND METHODS

In this study, 4 dogs were employed to determine the tolerance to the drug. All other dogs (153) were clinical cases. Chlorpromazine was administered orally in tablets of 10 and 25 mg. and parenterally in solution from ampules of 25 mg. per cubic centimeter.

TOLERANCE STUDIES WITH DOGS

In therapeutic doses, chlorpromazine is well tolerated by dogs. Courvoisier *et al.*[1] reported no deaths in dogs given 50 mg. per kilogram of body weight subcutaneously, or 100 mg./kg. orally. In this series of 113 dogs, no serious or undesirable side effects were encountered except in 1 case when a large dose was given intravenously at a rapid rate. The tolerance studies were as follows:

Case Reports.—A 16-year-old, 15-lb., mixed Terrier, deaf, blind, and belligerent, was given 25 mg. of chlorpromazine intramuscularly and in 20 minutes was depressed sufficiently to be handled and given 1 cc. (1 gr.) of pentobarbital intravenously without the aid of an assistant. Surgical anesthesia was produced. In three hours, the dog was able to lie on the sternum and in another 30 minutes was able to walk.

A healthy, 6-month-old, 30-lb., mixed Terrier was given 50 mg. of chlorpromazine intravenously. Immediate depression followed, with mild ataxia. In one and one-half hours, 4.6 cc. (4.8 gr.) of pentobarbital was given intravenously. Surgical anesthesia was produced for two hours, then the dog started swimming motions; 25 mg. of chlorpromazine given intramuscularly produced quiet sleep for six hours. The dog was able to stand upon awakening.

A mixed Terrier, 8 months old and weighing 25 lb., was given 125 mg. of chlorpromazine intravenously at a rapid rate. When three-fourths of the dose had been given, the dog stiffened, arched its back and neck, and urinated. The remaining one-fourth dose was given quickly and the dog became limp and was unable to stand for 20 minutes. In 30 minutes from the time of injection, it could walk and bark. The depression lasted several hours.

An emaciated, 10-year-old mixed Terrier was given 125 mg. of chlorpromazine subcutaneously. There was depression in 30 minutes which lasted about ten hours.

Experience based on these trials shows the drug to be safe at therapeutic doses.

CHLORPROMAZINE AS A PREANESTHETIC SEDATIVE

Group A.—Sixteen dogs, brought to the clinic for surgery, were given chlorpromazine, 1.5 mg. per pound, orally one hour before induction of anesthesia. The 16 operations included 12 oophorectomies, two enucleations of the eye, one perineal herniorrhaphy and castration, and one superficial tumor.

The pentobarbital was slowly administered intravenously. The excitement phase of induction usually encountered before the depressant action of pentobarbital sets in, was completely absent in all dogs to which chlorpromazine had been administered. In all instances, the recovery from anesthesia was completely devoid of delirium or restlessness until the sense of balance had been

Dr Estrada is a small animal practitioner in West Haven.

*Smith, Kline, and French Laboratories, Philadelphia, Pa.

restored. This is in accordance with the results obtained in man.[1-4]

Group B.—In 22 surgical cases chlorpromazine was administered intramuscularly at the rate of 1 mg. per pound of body weight 20 minutes before the induction of anesthesia. In all cases, the induction of anesthesia was smooth and awakening was devoid of delirium or restlessness. The operations carried out in this group were: 14 oophorectomies, 3 ear trimmings, three reduction of fractures; one castration; and one dental extraction.

Group C.—Eight dogs presented for minor surgery were administered chlorpromazine both intravenously and intramuscularly, followed immediately by pentothal. It did not appear that the chlorpromazine benefited the use of the pentothal.

Effect of Chlorpromazine on Nervous or Unfriendly Dogs

Das *et al.*[5] found that monkeys that had been given chlorpromazine were easily handled. This suggested the advisability of using the drug for unfriendly, nervous dogs. Six dogs were used for this purpose:

Case Reports.—A Cocker Spaniel, 32 lb., had eye surgery following intramuscular administration of 25 mg. of chlorpromazine. The dog continued to resist and bark, but a week later it was brought in for bathing and was given 50 mg. of chlorpromazine intramuscularly and in 20 minutes it could be handled satisfactorily, although it did bark hysterically three times during the two hours it was in the hospital. When the dog was dismissed two hours after administration of the drug, it was fully conscious and walked normally.

Four dogs, brought in for grooming and bathing, were difficult to handle and were given chlorpromazine intramuscularly at the rate of 2 mg. per pound. In each instance, the dog could be handled easily in 20 minutes after the drug was given. The effects lasted at least two hours.

A friendly, but barking, 20-lb. male dog left at the hospital was given 75 mg. of chlorpromazine subcutaneously. In 15 minutes, the dog became drowsy and slept throughout the night. He ate the following morning and no further disturbance was heard from him.

While the number of cases is small, the results indicate that chlorpromazine favorably affected the behavior of these dogs. A dose of 2 mg. per pound of body weight is suggested, but it may be necessary to increase the dosage in some instances. At 2 mg. per pound, a transitory ataxia may develop which usually disappears about 20 to 30 minutes after its onset.

Chlorpromazine as an Antiemetic

Authors have cited the usefulness of chlorpromazine as an antiemetic in human medicine and Brand *et al.*[6] in experimental work, have shown its usefulness in dogs and cats.

Chlorpromazine was used as an antiemetic in connection with the administration of an anthelmintic. It was used parenterally at the rate of 1 mg. per pound in 8 patients that were known to show persistent vomiting and orally in 46 patients that had vomited anthelmintics previously. The dose of chlorpromazine was 1 mg. per pound, followed in one hour by the anthelmintic. The same anthelmintic and dosage given previously was repeated and in all 69 patients vomiting of the anthelmintic did not occur. The drug did not interfere with the purging action of the anthelmintic. Chlorpromazine is now being given routinely to animals before they are given anthelmintics.

The remaining cases (11) consisted of dogs with various nervous conditions. Of these, only animals with senile agitation were benefited by the use of chlorpromazine.

Summary

A review of the pharmacodynamics of chlorpromazine suggests many clinical uses in veterinary medicine. The drug, in the experience of the author, when used in adequate doses, is superior to morphine, demerol,® or mephenesin to abolish postanesthetic delirium and restlessness from pentobarbital anesthesia. Its depressant action, when larger doses are employed, effects a marked influence on the behavior of nervous dogs and makes aggressive or unfriendly animals easier to manage without provoking loss of consciousness.

The antiemetic activity of chlorpromazine makes it invaluable in conditions associated with persistent emesis. When given orally, prior to the administration of anthelmintics, vomiting did not occur, yet there was no interference with the purgative action of the vermifuge.

ADDENDUM

Since this article was written, the number of animals on which we have used chlorpromazine has more than doubled and we have concluded that this drug is of definite value in treating certain neuroses in dogs. It appears to eliminate certain fears and, when given orally for a considerable period, unfriendly dogs acquire a more friendly attitude. "Shy" dogs also become less so.

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Clinical Uses for Chlorpromazine

Chlorpromazine hydrochloride, a phenothiazine derivative, is closely related to the antihistamine promethazine hydrochloride. It is a powerful central depressant, providing a general sedative effect differing from most sedatives in that consciousness is retained. It is a centrally acting anti-emetic and also produces vasodilatation and counteracts anoxia.

The intravenous injection of 2 mg./kg. in dogs immediately after severe hemorrhage protected them against shock which caused the death of 83 per cent of untreated controls. Healthy dogs have been given 5mg./kg. daily for 30 days with no side-effects and no histopathological changes.

Chlorpromazine should not be given while an animal is under the influence of an anesthetic or a narcotic or where liver or lung lesions exist. It should be given only intramuscularly to horses and cattle.

Cases are reported where it was injected intramuscularly to quiet refractive animals (after 40 minutes); intravenously, before a local anesthetic was given for surgery (after 5 to 8 minutes); *per os*, two to four hours before a journey, to prevent car sickness; and intramuscularly for heat stroke, tetanus, and spasmodic colic in horses.—*Vet. Rec., Dec. 3, 1955.*

Chlorpromazine May Cause Jaundice

In a study of jaundice development during chlorpromazine therapy, 43 cases were found, an incidence of about 1 per cent. It was more common in females but bore no relation to the dose and duration of therapy. It varied from mild to a severe, prolonged and, occasionally, a fatal illness. Chlorpromazine therapy should be avoided when infective hepatitis is endemic, in the presence of liver disease, in malnourished patients, when other hepatoxic agents are used, and when estrogens have been given. When jaundice occurs, chlorpromazine and all other drugs should be withdrawn and a high-protein, high-caloric diet given.—*J.Am.M.A., Jan. 14, 1956.*

Chlorpromazine of Benefit in Tetanus.—Muscular spasms can be relieved and anxiety replaced by "a lazy sleepiness" by the use of chlorpromazine. It has been used successfully, with a barbiturate and the antitoxin, in treating patients with tetanus.—*Sci. News Letter, Dec. 24, 1955.*

Effect of Chlorpromazine on Dogs

Chlorpromazine (thorazine) administered to dogs in doses of 10 mg. per kilogram of body weight produces stasis in the biliary tree, due to an increase in the resistance of the choledochoduodenal sphincter. These changes appear to be related to the depressing effects of chlorpromazine on duodenal motility.—*(Direct quote) Proc., Mayo Clinic, Dec. 14, 1955.*

Toxic Liver Dystrophy of Pigs

Toxic liver dystrophy was the chief cause of a 9 per cent mortality in baby pigs in Estonia in 1952. Intoxication from ingestion of moldy and decomposing foodstuffs such as cottonseed cake, and vegetables was considered as the chief cause, with a deficiency of vitamins A, B, or C as a predisposing cause.—*Vet. Bull., Dec., 1955.*

Lead Poisoning in a Dog

G. D. PETTIT, D.V.M.; L. W. HOLM, Ph.D;
W. E. RUSHWORTH, D.V.M.

Davis, California

On Jan. 1, 1955, an 11-month-old male Wire-Haired Fox Terrier, weighing 16 lb., was referred to the University of California veterinary clinic with a history of intermittent nervousness and anorexia extending over a period of two months. Symptomatic therapy failed to give relief and the day preceding admission the attack culminated in running and champing fits.

Pentobarbital sodium was used to control the nervous signs and upon recovery from anesthesia the dog appeared to be normal, although thin and slightly dehydrated. During the next eight days, the dog appeared normal. A fecal examination was negative for parasite ova and the urine was indicative of a mild cystitis, which subsequently abated.

In three blood samples taken during the first week of hospitalization, there was an increasing leukocytosis, a shift to the left, and many nucleated erythrocytes. On bone marrow aspiration a myeloid:erythroid ratio of 0.61:1.00 was found, a decrease from the 1.36:1.00 average ratio.[1]

Abdominal radiographs made on January 8 revealed the presence in the stomach of a square, flat, metal object (fig. 1). A lead drapery weight and rubber band were removed by gastrotomy the same day. The stomach was hyperemic, dilated, and atonic. Erosion patterns on the lead weight gave evidence that it had been present in the stomach for a considerable time. Because of the nature of the foreign body, blood and urine determinations for lead were made two days after surgery by the dithizone method as described by the Association of Official Agricultural Chemists. Lead was present in whole blood at a level of 0.6 mg. per liter and the urinary lead concentration was 0.7 mg. per liter; 0.08 mg. lead per liter is considered to be the maximal normal value for either blood or urine.[3]

DISCUSSION

Plumbism in dogs is uncommon.[2,3,9] A positive diagnosis of lead poisoning depends upon the occurrence of clinical signs together with the demonstration of abnormal absorption of lead.[3] However, Bond and Kubin[2] believe that when the hemogram is considered with appropriate signs, it is sufficiently characteristic to justify a diagnosis of lead poisoning. Lead assays, per se, are not considered absolutely necessary for diagnosis.

Lead poisoning in the dog manifests itself primarily as a gastroenteritis, with occasional nervous signs, anorexia, emesis, constipation or diarrhea, and colic. Deposition of lead sulfide along the dental margin of the gums to form the "lead line" is of diagnostic significance in man[3]; its occurrence is variable in horses and cattle.[5,7] No lead line was observed in the case reported here and it was not observed by Bond and Kubin[2] in their series of 10 dogs. It may not be a part of the syndrome in dogs. Anemia and basophilic stippling of erythrocytes are often stated to be among the most significant findings in cases of lead poisoning.[2,3] Bond and Kubin[2] consistently found anemia and stippling of normoblasts,

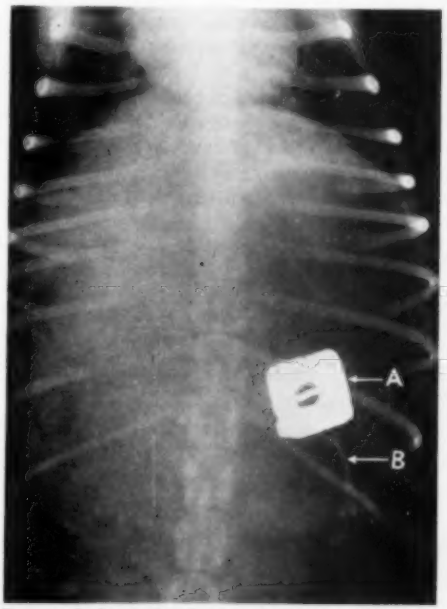

Fig. 1—Radiograph of dog showing lead drapery weight (A) and rubber band (B) in the region of stomach.

From the School of Veterinary Medicine, University of California, Davis. Dr. Rushworth was a senior student in veterinary medicine at the time of this study. He is now practicing in Palo Alto, Calif., with Dr. Robert C. Lawson, who referred the case.

TABLE 1—Hemogram During the Course of Chronic Lead Poisoning in a Dog

	Jan. 3	Jan. 5	Jan. 6	Jan. 14	Jan. 26	Jan. 29
Hemoglobin (Gm./100 cc.)	14.3	---	13.4	13.5	12.5	13.0
Erythrocytes (millions/cmm.)	7.45	---	6.75	6.37	6.72	7.15
Leukocytes (per cmm.)	15,400	25,500	34,600	28,400	20,300	23,400
Leukocytes (corrected for nucleated erythrocytes)	10,000	17,300	18,000	---	---	---
Nucleated erythrocytes/100 leukocytes	53	48	89	1	2	---
Packed cell volume	45	---	39	39	39	36
Lead in whole blood (mg./liter.)	---	---	---	1.04	---	---

but the stippling of mature erythrocytes was not always present.

The most striking findings in the hemogram of the case reported here (table 1) were the leukocytosis and the nucleated erythrocytes. These findings, although commonly reported in cases of plumbism,[3] are not pathognomonic. Basophilic stippling was not observed in any of the blood samples taken from the dog, nor was the animal markedly anemic.

TREATMENT

Despite recent success in treating lead poisoning in man,[4,10] horses,[5] and calves[6] with calcium disodium ethylenediaminetetraacetate (CaEDTA), there appear to be no records of its use in dogs.

CaEDTA therapy was initiated on January 10, two days after the lead weight had been removed from the stomach. A total dose of 0.5 Gm. was administered each day for four days. The initial 0.5 Gm. was added to 250 cc. of 5 per cent glucose in saline and was administered subcutaneously. Subsequent daily doses were divided: 0.25 Gm. added to 125 cc. of aminosol® was injected intravenously, while 0.25 Gm. diluted with 10 cc. of saline solution was given subcutaneously.

During the first three days of treatment, the patient neither ate nor defecated. He vomited daily for 19 days. Probantheline bromide, metropine, and phenobarbital were not markedly successful in controlling vomition. On the fourth day of treatment, the patient was hand-fed small amounts of canned dog food at frequent intervals. This dietary regimen was continued even though the appetite returned to normal. To combat the severe dehydration, the patient continued to receive 250 cc. of 5 per cent glucose in saline solution, plus injectable vitamin B complex, subcutaneously each day for another week.

TABLE 2—Urinary Lead Values of Dog During the Course of Treatment with CaEDTA

Date (Jan.)	CaEDTA dosage (Gm.)	Urinary lead (mg./liter) Morning (pre-treatment)	Afternoon (post-treatment)
10	0.5	0.7	---
11	0.5	---	7.2
12	0.5	---	21.1
13	0.5	---	26.0
18	---	0.2	---
20	0.5	0.5	30.2
21	0.5	2.4	---
22	0.5	7.2	22.3
23	---	5.7	23.6
24	---	0.5	---
26	0.5	3.0	45.3
27	0.5	0.45	12.5
28	0.5	0.15	2.9
29	---	0.31	0.63
30	---	0.79	---

A second series of CaEDTA treatments was begun seven days after the completion of the first therapy trial. It consisted of 0.5 Gm. of CaEDTA in 250 cc. of 5 per cent glucose in saline solution each day for three days. At this time, vomition was limited to one or two episodes per day, the bolus of food being re-eaten and retained.

After another four days, the third and final series of CaEDTA injections, identical to the second series was begun. At its termination on January 28, there was no vomition and the dog was alert, active, and gaining weight. He was discharged from the hospital on January 30, one month after admission. The urinary lead level (table 2) increased markedly within a few hours after the administration of the chelating agent. The concomitant diuresis, from the administration of large volumes of fluid, undoubtedly aided in flushing lead from the circulation. Since the total urine output could not be measured, we have no data on the total lead elimination. Also, because of the precarious condition of the animal, determinations of blood lead concentrations were not made.

SEQUELAE

On March 26, 1955, the dog was returned to the hospital. He had not progressed well at home and in the previous five days had deteriorated into a virtually moribund condition. Polydipsia, anorexia, and vomition had persisted during most of the convales-

cent period. The temperature was 105.0 F., pulse 200. A radiograph of the abdomen was taken because of the persistent vomiting and the film showed an irregular mass in the pyloric region. In the belief that this was a foreign body, another laparotomy was performed immediately. The gastrostomy wound had healed completely. The shadow in the film was thought to be that of the pancreas which was unusually large. At the termination of the laparotomy, cardiac arrest occurred, but the patient was revived with cardiac massage through the thoracic wall, the use of oxygen, and intracardial injection of 1.0 cc. of 1:1,000 epinephrine. Death occurred three hours later, the immediate cause being hemopericardium.

TABLE 3—Postmortem Lead Levels in a Dog with Lead Poisoning

Blood	1.0	mg./liter
Urine	0.125	mg./liter
Bone (skull)	4.15	mg./100 Gm. (dry weight)
Kidney	1.7	mg./100 Gm.
Liver	0.205	mg./100 Gm.

Postmortem examination of the tissues showed gross and histological nephritis and excessive amounts of lead in the blood, urine, kidney, liver, and bone (table 3).

SUMMARY AND CONCLUSION

A case of lead poisoning in a dog including temporarily successful treatment with CaEDTA, is reported. This case illustrates that a large amount of lead can be absorbed and deposited over a relatively short time from a solid lead object present in the stomach. It also illustrates the great mobilization potential of CaEDTA with respect to lead in a naturally occurring toxicosis in a dog. The unfortunate relapse and ultimate death of the patient was probably due to the premature cessation of therapy when laboratory evidence indicated substantial amounts of lead were still being excreted. In retrospect, giving full consideration to the postmortem lead level in bone depots and soft tissues, it is felt that subsequent dietary management of the patient should have included intensive calcium treatment, by either oral or parenteral administration.

Although the immediate cause of death may be ascribed to hemopericardium which resulted from the intracardial injection of epinephrine, the underlying factors are difficult to assess. Tissue lead levels and the history of the patient implicate lead toxicosis as a major factor contributing to the death. The extent to which the subacute interstitial nephritis complicated the toxicosis remains speculative.

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Western Equine Encephalitis in East

Western equine encephalitis virus was isolated from North Carolina mosquitoes after a recent epizootic of that disease. It was found in engorged *Culex quinquefasciatus* and represents the second isolation of this virus on the eastern seaboard, the first being in New Jersey in 1954. Eastern equine encephalitis virus was also recently isolated from horses and pheasants in North Carolina. The presence of both types of virus will make investigations of the disease difficult.—*U.S.P.H.S. Release, Dec. 22, 1955.*

Several types of cancer of man can be grown in laboratory animals, in test tubes, and in fertilized eggs.—*Sci. News Letter, Dec. 31, 1955.*

Disease Conditions Observed in Lambs at Slaughter

W. D. POUNDEN, D.V.M., Ph.D.; D. S. BELL, M.S.;
B. H. EDGINGTON, D.V.M.; D. L. THOMAS, D.V.M.

Wooster, Ohio

OHIO IS ONE of the leading sheep-producing states but little information is available concerning losses from disease conditions in animals from this state, including those problems which only become apparent when lambs are inspected after slaughter. The present investigation was undertaken to obtain information on the incidence and extent of the various lesions encountered at slaughter for purposes of future research planning and to help in directing disease control programs.

Sheep often are subject to two different types of postslaughter inspection: (1) to eliminate from sale any portions of the slaughtered animal deemed unfit for human consumption, and (2) to meet the requirements of the kosher trade. Kosher inspection may result in complete rejection of a carcass due to conditions not recognized as sufficient for condemnation by the former type of inspection, e. g., the presence of fibrous adhesions and scar tissue involving the pleura.

Failure to pass either of these inspections results in financial loss.When a lamb passes usual health inspections but fails to be accepted for kosher trade it sometimes means a loss of as much as $2.00 or more in net returns. Besides the economic losses related to the carcass, others may be associated with by-products such as casings. Nodular worm infection, for example, may damage the small intestines and render them unsuitable for the more desirable commercial outlets. The value of an acceptable small intestine is approximately $1.50 per lamb. Parasite lesions or abscesses in livers and lungs may also add to the economic losses that occur because of unhealthy conditions in slaughtered lambs.

From the Ohio Agricultural Experiment Station, Wooster.

Assistance was provided in the development and conduct of this study by R. H. Grimshaw, Agricultural Extension Service; J. K. McCarey, F. Barnett, and S. Oliver, Swift and Company; G. Lane, Producers Livestock Commission Association; other members of the Departments of Veterinary Science and Animal Science, Ohio Agricultural Experiment Station; and U.S.D.A. inspectors at Cleveland, Ohio. This assistance is gratefully acknowledged.

EXPERIMENTAL PROCEDURES

When ready for market, groups of 9 to 21 lambs varying in on-foot grade from low good to prime were chosen from 28 different farm flocks in Knox County, Ohio. The total number of lambs was 331. On seven different occasions four groups each time were followed through separately to postslaughter inspection at Swift and Company, Cleveland, Ohio. These examinations were made on Sept. 28, Oct. 12 and 26, Nov. 9 and 23, 1954, Jan. 18 and March 8, 1955. In addition, three fairly equal groups, totaling 88 lambs, from the flock of the Ohio Agricultural Experiment Station, Wooster, were examined on Oct. 26, Nov. 23, 1954, and Feb. 1, 1955. It was also possible to examine an additional 72 lambs of unknown origin slaughtered on Feb. 1, 1955, and 50 on March 8, 1955. It was thought these 122 lambs came from neighboring states.

RESULTS

A summary of the thoracic lesions is presented in table 1 and a summary of the abdominal lesions in table 2.

In the tables, opposite each examination date, are listed the total number of lambs inspected from four of the 28 farm flocks, or the number of lambs from the Ohio Agricultural Experiment Station flocks, or the number of unknown origin. Listed separately in table 1 are the number of lambs with pleural adhesions, readily detectable pneumonic lesions which were chronic in nature, lesions suspicious of the same condition, and lesions of the type that are associated with the lung parasite *Muellerius capillaris*. Pleural adhesions were accompanied by lesions in the underlying lung tissues in the vast majority of the cases. Most of the lesions in the lungs appeared to have been present for extended periods as indicated by the formation of scar tissue.

Included in table 2 are the total number of lambs affected with nodular lesions in the intestines, the number of commercially unacceptable small intestines, and the number of livers affected with abscesses. To be commercially acceptable, the small intestines had to be practically free of nodules. It is doubtful if any were counted as

acceptable that had more than 2 or 3 nodules in their entire length.

Occasionally, small fibrous lesions of undetermined origin were seen in livers but these were not included in the table.

THORACIC CAVITY LESIONS

Pleural adhesions were present in slightly more than 7.0 per cent of the combined total of 541 lambs and the incidence varied little between groups. Somewhat more than twice this percentage had definite pneumonic lesions and approximately double the latter had suspicious lesions.The net result was that nearly half of the lambs had lesions indicative of previous active thoracic disease. A seasonal trend appeared to exist as the percentage of pulmonary lesions showed a rise from the September examination on into November. This was followed by a downward trend to the last examination conducted (March 8, 1955). The incidence of lesions which appeared to be the result of parasitism by *M. capillaris* averaged 60.3 per cent for the lambs of known origin. The record was incomplete on the lambs of unknown origin, so figures for these were omitted. An estimate however, indicated rather similar incidence of infection in these lambs.

ABDOMINAL CAVITY LESIONS

Although nodular lesions were detected in the intestines of 52.8 per cent of the lambs of known origin from farm flock samples, only slightly more than 9.0 per cent of the small intestines had sufficient lesions to be unacceptable for better trade purposes. The latter figure was even smaller when the lambs of unknown origin were included (table 2). Lambs from the Ohio Agricultural Experiment Station flocks were free of nodular lesions in both large and small intestines at all three examinations. This was true of only one other flock sample. However, the lambs from 14 other flocks were almost free as there were no unacceptable small intestines among them. In six additional flock samples, not more than 1 lamb in 10 was sufficiently affected to cause rejection of the small intestines.

Seasonal trends were apparent in Knox County lambs, both in the numbers of unacceptable small intestines and in the presence of nodular lesions in either large or small intestines. Lowest percentages were recorded in September and October, and the highest ones at the time of the later examinations in 1955.

Liver abscesses were not found, to any degree, until the January 18 examination at which time 5 of the 6 lambs found so affected were all from one flock. The overall incidence was only 12.4 per cent for farm flocks of known origin but on the last examination (March 8), the incidence was almost 60.0 per cent and many of the lesions were extensive. Among lambs from flocks of unknown origin, liver abscesses were numerous at the time of the examination on

TABLE 1—Pathological Conditions Observed in the Thoracic Cavity of Lambs at Slaughter

Examination date	Lambs (No.)	Pleural adhesions	Pneumonic lesions				Parasitic (M. capillaris) lesions
			Definite	Slight and suspicious	Total	Percentage	
28 Farm Flock Samples (Knox Co.)							
9/28/54	50	7	8	5	13	26.0	37
10/12/54	48	2	5	23	28	58.3	42
10/26/54	44	4	7	15	22	50.0	15
11/9/54	42	1	10	23	33	78.5	31
11/23/54	46	3	15	18	33	71.7	28
1/18/55	44	2	5	22	27	61.4	42
3/8/55	57	4	4	12	16	28.0	29
Total	331	23	54	118	172	52.0	224
O. A. E. S. Flock							
10/26/55	25	4	8	12	20	80.0	10
11/23/55	34	3	3	12	15	44.1	13
2/1/55	29	1	2	8	10	34.5	6
Total	88	8	13	32	45	51.1	29
Origin Unknown							
2/1/55	72	9	13	14	27	37.5	No record
3/8/55	50	0	6	5	11	22.0	No record
Total	122	9	19	19	38	31.1	No record
Grand total	541	40	86	169	255	.....	253[1]
Percentage	.....	7.4	15.9	31.2	47.1	.....	60.3[1]

[1]Based on the total of 419 lambs of known origin.

TABLE 2—Pathological Conditions Observed in the Abdominal Cavity of Lambs at Slaughter

Date	Lambs (No.)	Nodular worm lesions	Commercially unacceptable small intestines	Liver abscesses
28 Farm Flock Samples				
9/28/54	50	6	2	0
10/12/54	48	24	3	0
10/26/54	44	11	3	2
11/9/54	42	30	4	0
11/23/54	46	30	5	0
1/18/55	44	28	4	6
3/8/55	57	46	9	33
Total	331	175 (52.8%)	30 (9.1%)	41 (12.4%)
O. A. E. S. Flock				
10/26/54	25	0	0	0
11/23/54	34	0	0	0
2/1/55	29	0	0	5
Total	88	0	0	5 (5.6%)
Origin Unknown				
2/1/55	72	No record	0	27
3/8/55	50	No record	4	7
Total	122	No record	4 (3.2%)	34 (27.8%)
Grand total	541	.....	34 (6.3%)	80 (16.6%)

February 1, but much less so among those examined on March 8.

DISCUSSION

The high incidence of lesions in the thoracic cavity was not expected. Whether it reflects a common condition or merely particular conditions that existed during the season concerned will need further study. Most of the lesions seemed to have been in process of healing for extended periods. This may help explain why the relative proportion of definite lesions to suspicious ones was higher in September than later. Some lesions which might have been definite if they could have been observed in September were so nearly healed by slaughter time at a later date that they were classified as suspicious. There did not appear to be any relationship between the presence of *M. capillaris* lesions and the pneumonic lesions.

In spite of the relatively high proportion of lambs that had some nodular worm lesions, the number of small intestines that were acceptable for better trade uses was high, about 94.0 per cent. This may mean that fairly persistent use of preventive measures, including phenothiazine-therapy, is having a favorable effect since all but one of the 28 farm flocks were receiving this drug. In further support of this, it may be mentioned that the Ohio Agricultural Experiment Station lambs have received commonly recommended phenothiazine treatments consistently for seven years and no nodular lesions were encountered in any of the 88 lambs from these flocks. The gradual trend of increasing incidence of nodular infections and unacceptable small intestines as the season progressed is similar to previous observations made in this state by others.[1]

A satisfactory explanation for the presence of the liver abscesses found in January-March period was not obtained. On the basis of work with feedlot cattle,[2] rumenitis would be suspected as the lesion which would permit portal entry of infection, portal entry being the one route by which the lesions were produced.[3] While rumenitis may have been a factor which entered the picture when the lambs first went on heavy grain feeding, lesions were not discernible in rumens at the time of slaughter except in animals from one flock and an occasional lamb in others. Lambs from this one flock were the earliest ones in the season to be found affected with liver abscesses.

Nodular worm lesions would seem to be a logical portal of entry for infections reaching the liver. Of the 46 lambs with liver abscesses, 34 had nodules in the intestines. However, there were 5 lambs in each of two flocks, a total of 10 animals, that were affected with liver abscesses but were free of nodular worm lesions. It is of course possible that both rumenitis and nodular lesions may provide satisfactory pathways for entry of liver infections. Possibly, some factors must exist other than the mere presence of means for entry of infection, such as nodular lesions or rumenitis, in order to permit the formation of liver abscesses. Support for this comes from the finding of nodular lesions in lambs on all examination dates including one in September, yet readily noticeable liver abscesses were not encountered until late in the marketing season and after lambs had been on winter feed for several weeks. Another factor to be considered would be the possibility of infections which differed from those observed by others[3] being responsible for the abscesses in the lambs in the present study. No check of this was made.

SUMMARY

During the period between September and March, sample groups of lambs from 28 farm flocks in Ohio, totaling 331 lambs,

along with 88 lambs from the Ohio Agricultural Experiment Station flocks and 122 from unknown sources were examined postslaughter for disease conditions, particularly those which would affect financial returns.

Pleural adhesions were present in 7.4 per cent of the lambs examined. Definite lesions of chronic pneumonia were found in 15.9 per cent and enough others had lesions suspicious of this condition to bring the total affected up to 47.0 per cent of the lambs.

Nodular lesions were detected in approximately 50.0 per cent of the lambs of known origin from farm flocks but only slightly more than 9.0 per cent of the small intestines had sufficient lesions to render them unsuited for top commercial uses. Lambs from the Ohio Agricultural Experiment Station flocks were entirely free of nodular lesions. These flocks had been on a parasite-preventive program consistently for seven years and presumably this played a major role. Only one other flock sample was entirely free of nodular lesions. However, all small intestines were acceptable for top commercial uses from half the flock samples and the records of several other flocks were almost as satisfactory as this.

Liver abscesses were found in approximately 16.0 per cent of the lambs but were not encountered to any degree until January, which suggests a connection with drylot feeding.

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[2]Jensen, R., Deane, H. M., Cooper, L. J., Miller, V. A., and Graham, W. R.: The Rumenitis-Liver Abscess Complex in Beef Cattle, Am. J. Vet. Res., *15*, (1954): 202-216.

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Virus Pneumonia Found in Swine

A virus pneumonia similar to that commonly found in swine in Britain has been identified in American swine. Dr. A. O. Betts, on leave from the University of Cambridge, England, states that pigs may remain carriers since the virus persists in the lungs for many months. The pigs usually cough and are unthrifty but do not appear ill. Since pigs seem to develop no immunity, chances for a satisfactory vaccine are small.—*Cornell Univ. Release, Jan. 5, 1956.*

Edema Disease Passaged in Rabbits

By subcutaneous injection of 10 per cent suspensions in saline of brain and cord from pigs believed to have died from enterotoxemia (edema disease), the authors claimed to have five times passaged the infection through rabbits. The rabbits developed paralysis of the forelimbs and soon died, without lesions or bacterial infection. However, no attempt was made to reproduce the disease in pigs.—*Vet. Bull., Dec., 1955.*

Equine Infectious Anemia Diagnosis.—Siderocytes (erythrocytes containing nonhemoglobin iron) were found in the blood of 96 of 142 horses with equine infectious anemia but never in uninfected horses. They could be a useful aid in diagnosis.—*Vet. Bull., Dec., 1955.*

Intradermal Tuberculin Tests in Swine

For tuberculin testing herds of swine in Europe, bovine-type tuberculin was injected intradermally in the back 10 to 20 cm. behind the shoulders and to one side of the midline. A positive, 72-hour reaction consisted of redness, thickening, and crust formation 25 to 50 mm. in diameter. Necropsies confirmed the value of the test.—*Vet. Bull., Dec., 1955.*

Chlorpromazine Dermatitis in Man

Dermatitis developed in 13 of 427 nurses and in 1 of 6 pharmacists at one hospital as a result of handling chlorpromazine in tablet or syrup form. The skin of the face, neck, and between the fingers was swollen, erythematous, and pruritic. Sensitivity was so great that contact with perspiration or saliva of a patient taking the drug often precipitated an attack. Antihistaminics were of little value but hydrocortisone ointment helped to allay the severe inflammation.—*J.Am.M.A., Jan. 7, 1956.*

Hereditary Deafness in a Family of Foxhounds

E. W. ADAMS, D.V.M.

Tuskegee Institute, Alabama

Best and Taylor,[1] in their classic report on the etiology of defective hearing, classify deafness as transmission, perceptive, and central. (1) Transmission deafness arises as a result of some interference to the passage of sound waves to the inner ear. It may be caused by a foreign body or wax in the external auditory meatus, inflammation of the middle ear (acute or chronic), or otosclerosis which would result in limitation of movement of the ossicles, causing the stapes to become fixed in the oval window. Inflammatory or neoplastic tissue at this location would also result in some degree of deafness. (2) Perceptive deafness results from developmental arrest or any developmental defect of the inner ear. This condition is most often congenital and the degree of deafness may vary from only slight impairment to deaf-mutism. (3) Deafness resulting from a lesion of the auditory pathways or of the auditory center is termed central deafness.

In Lindenov's[3] study of hereditary deafness in man, he found perceptive deafness to be associated with developmental anomalies of the inner ear. He reported that the chief pathologic-anatomical change in ten persons with congenital deafness was malformation of the bone of the cochlea in association with defects of the nerve tissue and malformation of the membranous connective tissue.

A review of the literature reveals that congenital deafness in dogs appears to be associated with lack of pigment, as the dogs which are affected are invariably white or have extensive white markings. According to Young,[6] genetic deafness is most common in Dalmatians, Bull Terriers, Sealyhams, Scotch Terriers, Border Collies, and Fox Terriers. Muller and Glass[4] commonly observed deafness in certain strains of pure white dogs such as Bull Terriers, which, on necropsy, generally showed atrophy of the auditory nerve.

Winge[5] has reported that when 2 typical "harlequin" Norweigan Dunkerhounds are mated, 25 per cent of their offspring will be white with small, gray spots, and this group will be totally deaf. Brumley[2] believes the deafness that is seen in young puppies is due to improper development of the auditory nerve or other structures of the internal ear.

CASE REPORT

A 1-year-old, tan-and-white Walker Foxhound from a kennel in which deafness was observed in certain bloodlines over a period of 14 years was referred to our clinic. Upon microscopic examination of its inner ear, abnormalities were found similar to those described by Lindenov.[3]

To maintain certain desirable characteristics, line breeding had been commonly practiced and as a result nearly all of these hounds showed some white markings. The yearly dog population had averaged about 150, with approximately 20 litters annually, of which 2 or 3 of the puppies were totally deaf. Deafness was first observed when the pups were 4 to 6 months old. They would show unresponsiveness and pay no attention to noises that caused their litter mates to prick up their ears and commence to bark.

Clinical examination revealed no gross pathological changes of the external ear,

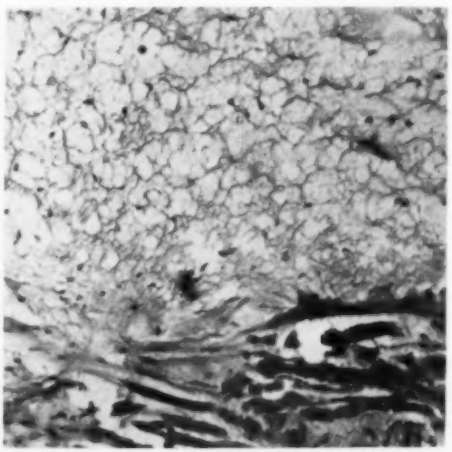

Fig. 1—Microscopic view of bulbous growth separating stapes from oval window, showing branched connective tissue cells scattered through the mucoid matrix. x 107.

Dr. Adams is assistant professor, Department of Pathology and Parasitology, School of Veterinary Medicine, Tuskegee Institute, Ala.

The author is indebted to Dr. H. Grady Young, a practitioner in Thomasville, Ga., for his interest in this case. Appreciation is expressed to Mr. C. P. Graham, medical photographer, Veteran's Administration Hospital, Tuskegee, Ala., for his collaboration.

Fig. 2—Microscopic section of cochlea of the Foxhound showing: (1) skinlike tissue in area normally occupied by structures essential to hearing; and (2) petrous temporal bone made up of layers. Note the normal cone-shaped appearance of this area which represents the cochlea. x 107.

but the signs were classical for a deaf animal. The expression was anxious and the ears frequently elevated at their base and pulled backward as if trying to hear. After euthanasia, the necropsy was negative except for a bulbous growth 5 mm. in diameter that enveloped the stapes, effecting a complete separation of this bone from the oval window.

The inner ear, brain stem, and the auditory nerve were removed intact. All tissues, including the bulbous growth, were fixed in formalin, sectioned, stained with hematoxylin and eosin, and examined microscopically. The bulbous growth separating the stapes from the oval window was found to be a myxoma (fig. 1). No pathological changes were apparent in those microscopic sections made of the auditory nerve or the brain stem. The microscopic anatomy of the inner ear was striking. The bone immediately surrounding the area in which the cochlea normally lies, appeared to be made up of layers, which suggested new bone formation. The only resemblance to a normal cochlea was the cone shaped case (fig. 2) which normally contains the structures essential to hearing. The organ of Corti, scala tympani, scala vestibuli, and other structures normally found in this area were absent. The area was occupied by tissue which was histologically similar to skin, complete with sweat glands, hair follicles, and sebaceous glands resting in a dense connective stroma.

SUMMARY

A case of congenital deafness is reported in which the etiological factor was found to be agenesis of the cochlea and a myxoma that completely separated the stapes from the oval window. Under the classification of Best and Taylor,[1] this would be designated as perceptive and transmission deafness.

Deafness had affected certain bloodlines at this kennel for 14 years. It appeared that heredity was a factor. Lesions similar to those described may well have been the cause of all this deafness.

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[5]Winge, O.: Inheritance in Dogs with Special Reference to Hunting Breeds. Comstock Publishing Co., Ithaca, N. Y., 1950.

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Leptospira icterohaemorrhagiae infection in calves recently occurred in Ireland. Two calves, less than 2 months old, died after a few days of illness, the chief lesion in 1 being a generalized jaundice. Urine and blood from this calf, injected intraperitoneally into 2 guinea pigs, resulted in their death nine days later, with Leptospira in their tissues.

Blood samples were taken from 6 other calves on the farm, only 1 of which had shown clinical illness, and 5 of the 6 were positive for *L. icterohaemorrhagiae* in titers of 1:30 to 1:1,000. All were negative to *L. pomona, L. canicola, L. grippotyphosa,* and *L. mitis.—Vet. Rec., Jan. 7, 1956.*

Intramuscular Cortef in Bovine Ketosis

J. ALLYN ROGERS, V.M.D.

Bryn Mawr, Pennsylvania

EXPERIMENTAL and clinical studies indicate that hydrocortisone, rather than cortisone, is the principal glycogenic steroid of the adrenal cortex. While the physiological activity of hydrocortisone is qualitatively similar to that of cortisone, it is quantitatively more active on a weight basis, its glucocorticoid activity being twice that of cortisone.

The chief metabolic effects include: (1) Increase in gluconeogenesis and deposition of liver glycogen, with decrease in carbohydrate utilization; (2) increase in urinary excretion of nitrogen and uric acid; (3) decrease in urinary excretion of sodium, chloride, and water associated with increase in urinary excretion of potassium, calcium, and phosphorus; and (4) increase in erythropoiesis with decrease in circulating eosinophils.

The hydrocortisone free alcohol used in the following studies was cortef,® sterile aqueous suspension, 50 mg. per cubic centimeter, which is intended for intramuscular injection for intense systemic hormonal effect. This is in contrast to the action of hydrocortisone acetate which is much less soluble and appears to have a slower rate of absorption from an intramuscular injection than the free alcohol form of hydrocortisone.

PART 1. INVESTIGATIONAL USE IN NORMAL COWS

First, to examine the magnitude of the metabolic effects of cortef in normal cattle, 5 healthy Guernsey cows, 3 to 8 years of age, were used. All were approximately in the middle of their lactation periods and were milking 10 to 18 lb. daily.

Prior to injection, 5 cc. oxalated blood samples were secured from each cow for blood sugar, ketone and hemoglobin determinations, red and white blood cell counts, differentials, and eosinophil counts. These details and the variations after intramuscular injection of cortef are presented (table 1).

Discussion.—The blood sugar average for the 5 cows before treatment was 43.6 mg./100 ml. All 5 responded to cortef injection with an elevation of blood sugar, increase in neutrophils, and decrease in eosinophils. There were no striking differences in animals injected with 30 cc. and 40 cc., as compared with those receiving 20 cc.

Cow "My.," which received repeated injections at 24 and 36 hours, plainly demonstrated elevation in blood sugar following each injection. A steady rise in neutrophils was also noticeable, and the eosinophils made but slight recovery in number. This would seem to indicate that this normal animal was capable of receiving successive stimulation with each injection of this hormone.

All animals demonstrated an attitude of well-being throughout and following the experiment. There was little change in milk production.

The intramuscular injection of cortef resulted in no untoward reactions in the doses used and demonstrated the desired metabolic effects for the treatment of bovine ketosis.

PART 2. TREATMENT OF FIELD CASES OF HYPOGLYCEMIA AND KETOSIS

Field cases of hypoglycemia and ketosis in cows were treated with intramuscular injections of cortef in the order in which they appeared in one practice.* All diagnoses were confirmed by blood sugar and blood ketone determinations performed in the laboratory on oxalated blood samples collected prior to therapy.

Two years of study have revealed that clients can be educated to place their calls early in the course of ketosis, rather than neglect the animals for the first two or three days of sickness. Thus, it becomes possible to treat nearly as many cases in the preketotic hypoglycemia phase as in the frank ketosis phase.

Project carried out while the author was assigned to ketosis study at the New Bolton Center, University of Pennsylvania, Kennett Square.

Cortef is Upjohn Company's trademark brand of hydrocortisone. Investigational quantities of cortef, S.A.S., 50 mg./cc., were supplied by the Veterinary Medical Department, Upjohn Co., Kalamazoo, Mich.

*The practice of Dr. James V. McCahon, Downingtown, Pa.

TABLE 1—Blood Changes After Intramuscular Injection of Cortef in 5 Normal Cows

Cows	RW			Ma			Va			Py			My		
Calved (born)	1/4/47			9/15/47			10/26/46			3/17/51			1/22/48		
Intramuscular dose	20 cc. (1,000 mg.)			20 cc. (1,000 mg.)			30 cc. (1,500 mg.)			40 cc. (2,000 mg.)			20 cc. (1,000 mg.) repeated at 24 and 36 hr.		
	Blood picture			Blood picture			Blood picture			Blood picture			Blood picture		
	Prior to injec.	24 hr. after	48 hr. after	Prior to injec.	24 hr. after	48 hr. after	Prior to injec.	24 hr. after	48 hr. after	Prior to injec.	24 hr. after	48 hr. after	72 hr. after	Prior to injec.	24 hr. after	36 hr. after	70 hr. after
Blood sugar (mg./100 cc.)	43	48	45	40	48	44	42	44	45	48	55	45	45	45	60	70	55
Blood ketones	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Red blood count (1,000)	5,620	5,900	6,190	5,690	5,720	5,590	6,290	6,700	7,750	6,500	7,240	5,850	5,320	5,830	5,560	5,440	5,950
White blood count	8,400	10,600	9,650	4,650	9,450	5,450	24,450	29,500	23,400	14,450	19,450	13,950	15,650	9,950	12,650	12,550	9,750
Neutrophil (%)	19	70	40	34	68	46	18	42	24	24	51	32	30	23	47	49	58
Lymphocyte (%)	50	22	31	56	36	43	73	54	68	64	42	55	47	54	46	40	34
Monocyte (%)	8	4	4	8	6	7	7	5	3	4	5	9	8	6	7	12	7
Eosinophil count	23	4	25	2	0	4	2	6	2	8	2	4	15	17	0	0	1
Eosinophil (%)	700	90	690	100	20	80	410	30	160	310	20	120	230	520	20	40	60
Hemoglobin (Gm.)	10.1	10.0	10.7	9.5	10.0	9.0	10.1	11.5	10.6	11.9	12.9	11.8	11.1	9.2	11.0	9.5	8.7

The hypoglycemic and ketotic animals studied (tables 2 and 3) were off-feed and down in milk production sufficiently to warrant the expenditure necessary to restore them to normal.

Discussion.—In 32 fresh cows, diagnosed as having preketotic hypoglycemia, the symptoms were inappetence and retarded milk production. Blood sugar determinations made on oxalated blood samples prior to treatment averaged 32.5 mg./100 ml. (compared with 43.6 mg. in 5 normal cows). There was no evidence of ketones in the blood. The average initial dose was about 555 mg. of cortef and 6 of 32 cows required a repeat treatment. All animals recovered.

TABLE 2—Bovine Hypoglycemia Treated with Intramuscular Cortef Injections

No. of cows	Initial dose (mg.)	Repeat dose (mg.)
4	1,000	.....
17	500	.....
5	250	.....
1	1,000	1,000
1	1,000	500
2	500	500
2	500	250

Total 32 Average initial dose, 554.7 mg./cow

In the 29 animals with clinical signs, diagnosed as having frank ketosis, the blood plasma gave typical ketone color reactions on Denco powder. The average blood sugar determination of this group was 30.0 mg./100 ml. All recovered and their milk production returned to normal. The average initial dose of cortef was about 690 mg., 24.3 per cent more than the animals in the early, or hypoglycemia, phase. Only 2 of 29 cows required a second treatment.

TABLE 3—Bovine Ketosis Treated with Intramuscular Cortef Injections

No. of cows	Initial dose (mg.)	Repeat dose (mg.)
14	1,000	.....
5	500	.....
8	250	.....
1	1,000	500
1	500	500, 500

Total 29 Average initial dose, 689.6 mg./cow

PART 3. PREVENTIVE USE IN A HERD OF HIGH-PRODUCING AYRSHIRES

The author has, for 30 years, been familiar with the history of a high-producing herd of Ayrshire cattle. Severe ketosis has

TABLE 4—Cows Prophylactically Injected Before and After Parturition with Cortef (50 mg./cc.)

Cow	History of ketosis	Dose cortef per injection	Data administered	Total cortef used	Date of parturition	Results
BK	None	150 mg.	9/4, 10, 15 20, 22, 25	900 mg.	9/29/54	No ketosis
HD	After previous parturition	150 mg. 250 mg. 150 mg.	11/5 11/23 12/1, 1/2, 7	850 mg.	11/29/54	Mild ketosis, good production
HF	None	150 mg. 100 mg.	9/10, 11 9/15, 25, 11/2	600 mg.	9/13/54	No clinical ketosis, but lab. evidence of blood ketones
HL	After previous parturition	150 mg.	4/18, 23, 29 5/6, 13	750 mg.	4/29/54 Twins, retained placenta, metritis, pyometra	No ketosis
Na	After previous parturition	150 mg.	11/5, 15, 26 12/6	600 mg.	11/17/54	Mild ketosis, good production
SP	None	100 mg. 125 mg. 150 mg. 100 mg.	9/4 9/8 9/10, 18 9/22, 25	725 mg.	9/8/54	No clinical ketosis but lab. evidence of blood ketones
		Ave. 143.7 mg. per injection	Ave. 5.1 injections per cow	Ave. 737.5 mg. per cow		

occurred repeatedly in some animals and approximately 50 per cent of the freshening cows have been affected. During the winter of 1953-1954, 15 cows developed clinical ketosis that required treatment.

A ketosis-preventive program was planned for the following year (1954-1955) as follows: Cortef, in small doses, was to be injected intramuscularly at periods two weeks prior to parturition, one week prior, at parturition, and once a week for two weeks following parturition. Six cows, several with known ketotic tendencies, were treated on this general plan (table 4). To prevent clinical ketosis with this method of administration required an average of 737.5 mg. of cortef per cow.

The necessity of the precalving injections was questioned, and to investigate the prevention of ketosis from another standpoint, 6 cows were injected only at varying intervals following parturition. These data are presented (table 5) and it is of interest that an average of 841.6 mg. of the drug was required for each cow.

In the same Ayrshire herd, 9 parturient cattle received no cortef therapy until physical symptoms of ketosis appeared (table 6). Intramuscular injections (150 mg. in most cases) were then begun and continued until milk production, appetite,

TABLE 5—Postparturient Cows Prophylactically Injected with Cortef (50 mg./cc.)

Cow	History of ketosis	Date of parturition	Ketotic	Dose cortef per injection	Date administered	Total cortef used	Results
LB	24 days after previous parturition	5/6/54	.....	150 mg.	5/10, 15, 20, 25, 30 6/4, 9, 14, 19, 24, 29 7/6	1,800 mg.	This amount of cortef required to keep cow in normal production
LG	None	2/18/55	.....	150 mg.	2/18, 21	300 mg.	No ketosis
He	3 days after previous parturition	1/13/55	.....	250 mg.	1/14, 16	500 mg.	No ketosis
Nk	None	1/16/55	Mildly	250 mg.	1/21, 27	500 mg.	No ketosis
WD	None	11/29/54	Mildly	100 mg. 150 mg. 250 mg.	10/30 11/1, 2 11/5 11/11	700 mg.	Mild ketosis throughout
Sb	7 days after previous parturition	1/12/55	.....	250 mg.	1/15, 24 2/3, 15, 26	1,250 mg.	No ketosis
				Ave. 180.3 mg. per injection	Ave. 4.6 injections per cow	Ave. 841.6 mg. per cow	

TABLE 6—Cows Receiving Cortef Injections After Early Signs of Ketosis

Cow	History of ketosis	Date of parturition	Ketotic	Dose cortef per Injection	Date administered	Total dose cortef
SA	None	3/8/54	8 days later	150 mg.	17 injections to 6/11	2,550 mg.
HB	6 days after previous parturition	3/13/54	7 days later	150 mg.	3 injections to 3/29	450 mg.
Bs	12 days after previous parturition	1/10/55	5 days later	150 mg.	3 injections to 2/22	450 mg.
BL	2 days after previous parturition	3/26/54	22 days later	150 mg.	4/18, 22	300 mg.
HC	None	12/12/54	10 days later	150 mg.	4 injections to 1/6	600 mg.
LD	None	3/30/54	4 days later	150 mg.	5 injections to 4/25	750 mg.
SN	None	12/23/54	47 days later	250 mg	2/9, 12, 14	750 mg.
PP	None	1/29/54	17 days later	150 mg.	2/15, 18, 21	450 mg.
RR	None	4/5/54	7 days later	150 mg.	4/12, 18, 29	450 mg.
				Ave. 157.2 mg. per injection	Ave. of 4.8 injections per cow	Ave. 750 mg. per cow

and attitude of the cows were satisfactory. With this method of treatment, each cow required an average of 750.0 mg.

Discussion.—Prevention or alleviation of ketosis in a susceptible herd is worthy of consideration when management and veterinary service can be integrated.

Cortef administered prophylactically to cows prior to and following parturition (table 4) resulted in continuous good milk production, although 3 of the 6 cows had been troublesome following previous parturitions. The data support the ketosis-preventive program of administering at least one injection of cortef prior to calving and two to four afterward to secure maximum efficiency at lowest cost.

Two to five postpartum injections (table 5) are worthwhile for prophylaxis, providing the first injection is made within the first one or two days.

Intramuscular injections of cortef easily controlled ketosis if begun after the earliest symptoms appeared. The average dose for recovery (750 mg.) closely approximated the total dose administered prophylactically.

It should be emphasized that professional service was required to treat clinical ketosis in 15 cows in this herd during the previous year. The cases were typical, with heavy loss in milk production. During the subsequent year, no professional emergency calls were necessary to treat the 21 cows tabulated in this study for ketosis. Close contact and consultation were maintained between the management and the veterinarian attending this Ayrshire herd.

The present study substantiates similar work with other herds and lends weight to the hypothesis that ketosis should be treated in the fresh cow rather than allowing it to develop into a complete metabolic breakdown.

CONCLUSIONS

1) Intramuscular injection of 1,000, 1,500, and 2,000 mg. of cortef, sterile aqueous suspension, 50 mg. per cc., to normal lactating dairy cattle produced an elevation of blood sugar and no untoward reactions.

2) Cows with hypoglycemia were relieved following intramuscular doses of 555 mg. of cortef.

3) Cows with clinical ketosis were relieved following intramuscular doses of 690 mg. of cortef.

4) When injections of cortef (720 to 850 mg.) in repeat doses of 150 to 200 mg. at three- to five-day intervals were initiated (1) before parturition, (2) at calving, or (3) when the first signs of ketosis appeared, signs of ketosis did not develop in susceptible, high-producing dairy cows.

5) The additional amounts of cortef required for prophylactic therapy (repeat injections of 150 to 200 mg. to a total dose of 720 to 850 mg.) would seem to be justified by the continued high level of milk production and absence of physical impairment (ketosis) to the cow.

Blackleg in Pigs.—Clostridium chauvoei infection caused the death of 34 pigs in Italy, 15 within two days. Edema of the pharyngeal region and degenerative myocarditis were constant.—*Vet. Bull., Dec., 1955.*

Sporadic Bovine Encephalomyelitis—Isolation and Antibiotic Susceptibility of a Texas Strain

D. A. PRICE, D.V.M., and W. T. HARDY, D.V.M.

College Station, Texas

SPORADIC BOVINE encephalomyelitis, a virus disease of calves, is commonly called "brain fever" by Texas ranchmen. First described in Iowa by McNutt[1] in 1940, and then by Boughton[2] in Texas in 1941, it has become well established in this state.

During the early 1940's, research at the Sonora substation confirmed the infectious nature of the disease, disclosed that guinea pigs were readily infected, demonstrated the presence of the agent in most body fluids and excretions, and indicated that infection tended to induce immunity in calves. More recently, workers in other states[3-9] have made important contributions to the knowledge of the disease. It is now believed to exist in at least nine states.

Our purpose here is to report the progress made at our laboratory since work was resumed on this disease late in 1954, and to review some of the previous knowledge.

DESCRIPTION OF THE DISEASE IN TEXAS

Records at our laboratory indicate that "brain fever" is rather well distributed in west-central Texas counties. However, it is probably distributed throughout much of the state. Its sporadic nature poses some interesting problems. The cattle on ranches adjoining an infected herd commonly are unaffected. Too, the infection may disappear from a herd spontaneously only to reappear several years later. In Texas, the disease is essentially similar to that reported from other states, except that it may be more mild. The mortality is less than 10 per cent of the calves affected.

The most common symptoms are: loss of appetite; gaunt appearance, with rapid loss of weight; rough coat and droopy ears; serocatarrhal ocular and nasal discharge; constipation, especially in older calves; locomotor ataxia (loss of muscular coordination) often followed by prostration; labored breathing; and, rarely, diarrhea, lameness, extended head and neck, and convulsive seizures.

Necropsy commonly reveals fibrinous peritonitis, pleuritis, and pericarditis; scaly or peeled muzzle; and congestion of the blood vessels in the brain covering. Arthritis is sometimes present.

ISOLATION OF THE CAUSATIVE AGENT

The tissues used were from an infected calf which had been sent to our laboratory for necropsy and diagnosis. A 50 per cent emulsion of liver, spleen and kidney in saline-glycerin solution was treated with streptomycin and used to inoculate 7-day embryonating chicken eggs via the yolk sac. Of 10 embryos inoculated, 4 died ten days later and 4 on the twelfth day. Serial egg passages were made with infected yolk material diluted equal parts with sterile broth or saline-glycerin. The agent apparently had become fully adapted to chicken embryos at the third egg passage, when all of 10 embryos were dead on the fourth day. At the present time, it has been stabilized at the twentieth egg passage level.

Infected yolk material was infectious for both guinea pigs and a calf. Symptoms and lesions produced were typical of natural infection.

DETERMINING THE INFECTIVITY

It is important to know the degree of infectivity of a disease-producing agent. Since egg embryo deaths usually occur following inoculation of the bovine encephalomyelitis agent, it was appropriate to determine the dose proving lethal to 50 per cent of the test embryos ($l.d._{50}$). Starting with 100 per cent infected yolk, tenfold serial dilutions were made with sterile beef heart infusion broth. Groups of 6 or more 7-day embryonating eggs were inoculated via the yolk sac route with 0.2 ml. of this

Associate veterinarian (Price), superintendent and veterinarian (Hardy), Ranch Experiment Station, Sonora, Texas.

Some of the intravenous ilotycin was furnished by Dr. G. T. Edds of Fort Dodge Laboratories. Both intravenous and intramuscular ilotycin were supplied through courtesy of Dr. F. O. Gossett of Eli Lilly & Co. Magnamycin and terramycin were furnished by Dr. G. E. Hawley of Chas. Pfizer and Co. Aureomycin and intravenous achromycin were supplied by Dr. H. R. Cox of Lederle Laboratories.

inoculum. Incubation was continued for seven days, the eggs being candled twice daily.

The embryo death pattern for a typical experiment is given (table 1). Using the

TABLE 1—Death Pattern for a Typical $l.d._{50}$ Determination

	Dilution of infected yolk						
	10^{-2}	10^{-3}	10^{-4}	10^{-5}	10^{-6}	10^{-7}	10^{-8}
No. embryos dead out of 8	8	8	8	8	7	6	5
Ave. postinoculation day of death	5.0	5.5	5.6	5.9	6.8	7.3	7.5

Seven-day embryos were inoculated via the yolk sac route with infected yolk from the thirteenth egg passage. Average day of death = the number of deaths occurring on each day multiplied by that day and the sum total of these values divided by the number of embryos used.

Reed and Muench[9] method for determining the $l.d._{50}$, the average of several titrations was found to be a dilution of 1:1,500,000 ($10^{-6.8}$).

SUSCEPTIBILITY TO ANTIBIOTICS

The activity of several antibiotics against the bovine encephalomyelitis agent when demonstrated in eggs was investigated. Wenner *et al.*[4] had previously reported that while chlortetracycline (aureomycin®), oxytetracycline (terramycin®), and penicillin each exerted activity against the agent, chlortetracycline was superior, results with penicillin and oxytetracycline being irregular. Our investigations covered tetracycline, carbomycin, erythromycin, chlortetracycline, and penicillin.

Methods.—A measured amount of the antibiotic to be tested was diluted with a 1:100 (10^{-2}) aqueous emulsion of infected yolk so that a concentration of 25 mg. per milliliter was achieved. From this point, doubling dilutions were made with nontreated 10^{-2} emulsion so that the virus concentration in the inoculum would remain constant while the antibiotic concentration was halved each time. From each concentration, eggs were inoculated via the yolk sac route with 0.2-ml. doses. The maximum antibiotic dose rate was 5 mg. or 5,000 μg. per embryo. Dilutions were made with a 10^{-3} yolk emulsion because at that concentration of the virus, all of the controls die consistently between the third and sixth days.

Our procedure was not intended to provide an accurate basis for comparing the extent of activity of these antibiotics with one another. We can conclude, however, that they do exert decided activity against the bovine encephalomyelitis agent when studied in embryonating eggs.

Clinical Observations.—Guinea pigs were infected by inoculating 0.1 ml. of 50 per cent infected yolk, intraperitoneally. Groups of 2 were treated with an intramuscular form of erythromycin (ilotycin) from one to five days following inoculation. When the virus and antibiotic were administered simultaneously, infection apparently was prevented. When the antibiotic was delayed one or more days, infection became apparent but was quickly suppressed, the elevated body temperature returning to normal rapidly (within 12 hours).

In cooperation with a local ranchman, selected field cases were treated during the early febrile stage of the disease; 5 received the intramuscular form of erythromycin, three times at 24-hour intervals at the rate of 4 mg. per pound of body weight; and 6 received tetracycline, intra-

TABLE 2—Effect of Antiobiotics on a Strain of Bovine Encephalomyelitis Virus

	Test Results									
μg.	Chlortetracycline		Tetracycline		Erythromycin		Carbomycin		Pencillin	
per egg	No. eggs used	Ave. day of death	No. eggs used	Ave. day of death	No. eggs used	Ave. day of death	No. eggs used	Ave. day of death	No. eggs used	Ave. day of death
5,000.0	....	....	17	10.1	10	14.0	10	13.3	8	14.0
2,500.0	10	12.4	13	8.8	10	14.0	19	13.5	8	10.9
1,250.0	10	13.4	18	13.4	10	14.0	19	13.9	8	14.0
625.0	20	11.9	18	12.7	10	14.0	20	12.8	8	11.6
312.5	18	12.0	17	12.9	10	14.0	20	9.3	7	10.4
156.3	19	10.3	18	13.3	10	13.5	18	6.8	8	10.0
78.0	19	7.7	16	13.1	10	13.7	20	5.5	8	9.4
39.0	10	5.7	18	12.4	10	11.5	20	5.5	8	8.8
19.5	9	5.7	17	10.3	10	9.7	20	5.1	8	7.3
9.8	10	5.4	18	8.8	10	7.7	16	5.2	8	6.5
4.9	10	4.3	18	6.8	10	5.6	8	4.0	8	5.8
Controls	20	4.7	18	3.5	10	4.4	19	4.2	8	4.8

All embryos dying within 48 hours after inoculation were discarded and not included in test results on the assumption that such deaths were due to nonspecific causes such as trauma.

Average day of death = the number of deaths occurring each day was multiplied by that day and embryos that survived to hatching were, to simplify the statistical records, considered dead on the fourteenth day for purposes of calculation. The sum total of these values divided by the total number of embryos used was calculated as the value for the average day of death.

muscularly, at the rate of approximately 2 mg. per pound of body weight, followed by penicillin (approximately 2,000 units per pound, intramuscularly), on three successive days. The elevated body temperatures of all 11 calves returned to normal within 12 hours and the owner declared that they had regained their original "bloom" within ten days. Two untreated calves died. Previous experience suggested that penicillin alone is not consistently adequate in suppressing the infection.

While these observations are limited and not completely controlled, the response to treatment in these few instances has been so favorable that the use of these antibiotics, along with good nursing care, seems indicated, especially if an early diagnosis is obtained.

SUMMARY

Sporadic bovine encephalomyelitis, an infectious disease of calves, is well established in Texas where the ranchmen call it "brain fever." Typically, it is characterized by fever, rapid loss of weight, and muscular incoordination.

A Texas strain of the causative agent was isolated from a naturally-infected calf and has been adapted to chicken embryos.

Several antibiotics exerted activity against the virus when studied by means of infected chicken embryos. This report ascribes pronounced activity to tetracycline, erythromycin, chlortetracycline, carbomycin, and penicillin.

Clinical observations suggest that the infection in both calves and guinea pigs can be suppressed by antibiotic treatment.

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Serological Study of Shipping Fever

Before feeder cattle were shipped from Alberta and Saskatchewan to Ontario and Quebec in September to December, 1954, 2,093 animals were given commercial bacterin ten days to six weeks before shipment, 4,062 were given anti-Pasteurella serum one to four days before shipment, and about 3,255 served as controls. Of the bacterin-treated group, which were shipped first when shipping fever was less prevalent, 2.3 per cent were affected and 0.47 per cent died; of the serum-treated group, 6.5 per cent were affected and 0.86 per cent died; and of the control group, 5.4 per cent were affected and 0.92 per cent died.

Blood samples were collected from more than 10 per cent of the three groups immediately before treatment and again from the same animals after shipment. These serums were tested against three types of *Pasteurella multocida* and against *Pasteurella hemolyticus*.

Before shipment, the majority of the animals were negative to all four Pasteurella antigens. After shipment, serums from the serum-treated and control groups showed a marked increase in titer for *Past. multocida* type C and a greater increase for *Past. hemolyticus*. These organisms were also the ones most commonly isolated from the lungs of the animals that died. The results indicate exposure to these organisms during shipment.—*Canad. J. Comp. Med., Nov., 1955.*

What Is Your Diagnosis?

Because of the interest in veterinary radiology, the JOURNAL publishes this month, and will continue to do so for the next several issues, a case history and accompanying radiographs depicting a diagnostic problem.

Make your diagnosis from the picture below—then turn the page ▸

Figure 1

History.—A 7-year-old Hereford cow had developed progressively increasing dyspnea and had lost considerable weight during the previous six weeks. She was afebrile and had a slight bulging over the right maxillary sinus. No air was passed through the right nostril. A stomach tube was passed readily through the left nasal cavity but an obstruction prevented it from being passed through the right side. A dorsoventrad radiograph of the face was taken (fig. 1).

(Diagnosis and findings are reported on next page)

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Maxillary sinusitis (right).

Comment.—Note in figure 2 the deviation of nasal septum (A) and the area of radiopacity (B) medial to the right cheek teeth. A trephine opening, made at the low point of the maxillary sinus, released a quantity of pus which had been under considerable pressure. Breathing returned to nearly normal immediately. *Corynebacterium pyogenes* was cultured from the exudate.

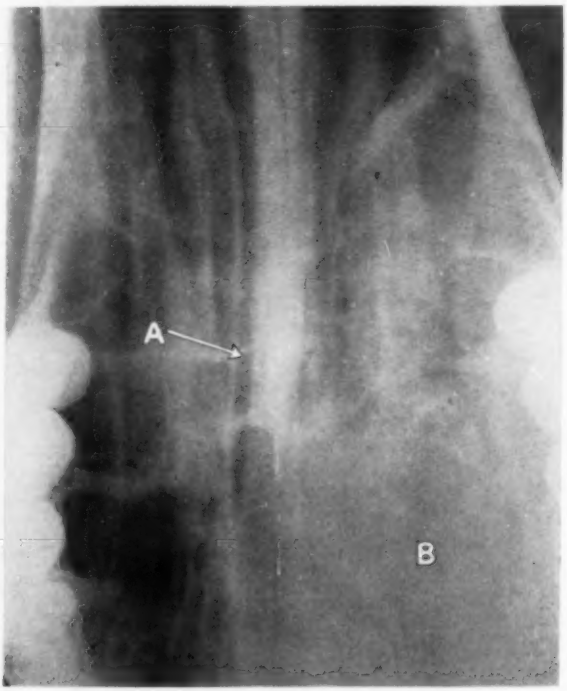

Figure 2

This case was submitted by Dr. William H. Beckenhauer, Department of Clinics and Surgery, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

Control of Egg-Transmitted Pleuropneumonia-like Organisms in Two Hatcheries Through Medication of the Foundation Stock

H. E. ADLER, D.V.M., Ph.D.; R. YAMAMOTO, M.A.; S. F. EXTROM, D.V.M.

Davis, California

PLEUROPNEUMONIA-LIKE organisms (PPLO) have been implicated as a major cause of chronic respiratory disease (CRD) of chickens and of infectious sinusitis of turkeys.

LITERATURE CITED

Egg transmission of infectious sinusitis of turkeys was demonstrated by Jerstad *et al.*[7] and later by Grumbles and Boney.[4] Van Roekel *et al.*[11] initiated the present interest in embryo transmission of CRD. They reported tracheal and other respiratory lesions, that were considered to be pathognomonic for CRD, in embryos that had been selected at random in commercial hatcheries and succeeded in isolating PPLO from these embryos. Cover and Waller[3] demonstrated the causative agent of CRD in a large percentage of pipped eggs, but they could not show a correlation between the presence of the organism in the eggs and the disease in the parent flock.

Fahey and Crawley[5] reported PPLO transmission by way of the egg in the epizoology of CRD. They considered that egg transmission played a major role in the dissemination of CRD.

Recently Jungherr *et al.*,[6] in a thorough study of serological testing for CRD, were able to demonstrate embryo transmission utilizing a number of tests, including hemagglutination-inhibition.

From the literature, it appears that PPLO have not been isolated from the genital tract of birds even though the chicks and eggs are later found to be infected with PPLO. In other animals, PPLO have been shown to be present in the genital tract but the role these organisms play in producing disease is not known. Nelson[9] demonstrated selective localization in the female genital tract of murine PPLO by intraperitoneal injection into mice.

The objective of this paper is to present the results of an attempt to control egg transmission of PPLO by medication of the foundation flock.

MATERIALS AND METHODS

Source of Birds.—The birds used in these experiments were obtained from two hatcheries designated A and B. Hatchery A had a flock of 240 first-year foundation White Leghorn hens and 24 roosters. Hatchery B had 1,100 White Leghorn hens and 76 roosters.

Several diagnoses of CRD had been made in birds from both hatcheries by culturing PPLO and by agglutination test from samples taken at intervals during an 18-month observation period. In almost every instance in which apparently latent CRD was detected by these means, it had been accentuated by either fowlpox vaccination or the actual occurrence of infectious bronchitis.

Treatment.—All of the foundation dams and sires in the two hatchery flocks were injected at two-week intervals with three doses of a mixture of streptomycin and dihydrostreptomycin sulfate* at approximately 25 mg. per kilogram of body weight. These birds also received 0.4 Gm. of erythromycin stearate† in the drinking water for the first five days of the treatment period.

Progeny Studied.—Prior to treatment, 200 blood samples were taken at random from one group of progeny from hatchery A and three groups of 200 each from hatchery B.

Following treatment, approximately 3,600 progeny at each hatchery (from the same breeders) were raised in separate buildings at the respective ranches.

In hatchery A, all birds were vaccinated twice intranasally with a combined Newcastle disease and infectious bronchitis vaccine at 1 day and at 10 weeks of age. Birds in hatchery B received a commercial coccidiosis vaccine in the feed at 3 days of age, intranasal Newcastle disease vaccine in the drinking water at 1 week, and laryngotracheitis and fowlpox vaccines at 8 weeks of age.

The PPLO Agglutination Test and Culture.—All serological tests were conducted with a modified PPLO plate antigen as previously described.[1]

Cultures for PPLO were made by the method of Adler, Yamamoto, and Bankowski.[2]

RESULTS

Two hundred blood samples taken at random from breeder hens in each hatchery were tested for PPLO agglutinins. Serum samples from hens in hatchery A showed 91 per cent reactors to the agglutination

From the School of Veterinary Medicine, University of California, Davis, (Adler and Yamamoto), and Practitioner (Extrom), Rio Linda, Calif.

*The streptomycin and dihydrostreptomycin were provided by Merck and Co., Rahway, N. J.

†Erythromycin stearate was provided by Abbott Laboratories, North Chicago, Ill.

test; those from hatchery B showed 93 per cent positive. Another 100 random blood samples from each group were taken two weeks after the last treatment and there was very little change in the reactor rate of the dams.

Blood samples taken from 200, 5-month-old pullets in hatchery A, hatched from eggs collected 14 days before medication, showed 6 per cent reactors to the agglutination test, whereas 4-month-old pullets hatched from eggs laid following the first treatment were negative to the agglutination test. The other two groups of pullets (viz. those hatched from eggs laid 10 and 20 days after the third treatment of dams) were negative to the test at 3 months of age.

Birds from hatchery B showed a somewhat different picture. Chickens hatched from eggs laid before the treatment had a reactor rate of only 10 per cent at 4.5 months of age. These birds were mildly affected with infectious bronchitis at 3 months of age. Two other groups of progeny from eggs laid before treatment, and which had a natural field occurrence of infectious bronchitis at 5 weeks of age, showed reactor rates of 60 and 80 per cent, respectively, at 3.5 months of age. During this experience with infectious bronchitis, PPLO were recovered from birds in both groups. In contrast, 200 progeny from eggs laid after the first treatment were negative to the agglutination test when 3 months old, and two other groups of 200 birds each, when tested at 6 weeks and 3 months of age, were also negative. Pleuropneumonia-like organisms were not isolated from the birds while infectious bronchitis was present (table 1).

TABLE 1—A Comparison of the PPLO-Agglutination Reactor Rate of Birds Hatched Before and After Medication of the Sires and Dams

	No. of birds	Age tested	Reactor rate*	Remarks
FLOCK A				
Progeny of untreated parents	200	5.0 mo.	6.0%	No respiratory disease occurred.
Progeny of treated parents	200	4.0 mo.	0	No respiratory disease occurred.
	200	3.0 mo.	0	
	200	3.0 mo.	0	
FLOCK B				
Progeny of untreated parents	200	4.5 mo.	10%	Clinically mild infectious bronchitis at 3 mo. of age.
	200	3.5 mo.	80%	Clinically severe infectious bronchitis at 5 weeks of age.
	200	3.5 mo.	60%	PPLO were recovered from 3 birds in each group.
Progeny of treated parents	200	3.0 mo.	0	Bronchitis at 3 weeks of age (see above);
	200	6.0 weeks	0	bronchitis at 3 weeks of age.

*Blood tests were conducted at one time within each flock.

When culled, the percentage of quality graded chicks** from hatchery A rose from 58.0 to 75.1 per cent within 32 days. Since all breeders were medicated in this flock, a control comparison was not made. Hatchery B had full sisters not on the treatment program for comparison. After three treatments, the percentage of quality chicks from the medicated dams was 80 per cent for the treated flock as compared with 65 per cent from the untreated, full-sister controls.

The 200 chicks from medicated dams and sires raised separately in strict isolation at the University of California and exposed to viral stresses remained nonreactors for ten weeks. No respiratory lesions were observed at necropsy and PPLO were not isolated from the tracheas of these birds.

Agglutinins were present in the serums of randomly selected, day-old chicks from the medicated hens. Twenty-five chicks 1 day old were destroyed in each group at intervals of five days to determine antibody in their serums. Agglutinins were present in 40 and 75 per cent of the serums of chicks from hatcheries A and B, respectively. These agglutinins were not measurable by this test beyond 15 days of age.

DISCUSSION

Field observations and limited laboratory trials implicate egg transmission as an important link in the perpetuation of CRD. Van Roekel and Olesiuk[11] showed that hatchability was significantly affected by CRD, and observed that the number of cull chicks was also increased. These observations were verified in the present study. Inapparent CRD occurred under field conditions and was aggravated into clinically obvious disease by infectious bronchitis virus infection. The almost explosive spread of PPLO at these times would appear to be due to the violent coughing incited by infectious bronchitis which, in turn, increases the disease-

**Final percentage of chicks saved after culling at 1 day of age.

spreading rate from PPLO-infected individuals.

Since the PPLO-agglutination test reactor rate in the dams was high, a treatment program was more practical than removal of these birds. While this paper was in progress, such a program was developed by Fahey and Crawley[5] who demonstrated that PPLO infection was prevented in progeny chickens by heavy medication of the dams and sires. Although the PPLO test procedure and drugs were different, the general program and results reported here were essentially the same.

Streptomycin and dihydrostreptomycin mixture, given by intramuscular injection, was selected for medication of the reactor sires and dams. The use of a combination of the two streptomycins was based on the work of Yamamoto[12] indicating differences in the *in vitro* and *in ovo* activity of these antibiotics against several different strains of PPLO of avian origin. Although *in ovo* and *in vitro* tests by Yamamoto indicated much greater activity against avian PPLO by the tetracycline drugs, these drugs were not selected because of the local tissue irritation they caused when injected. Smith[10] showed that injection of streptomycin sulfate in chickens produced higher blood levels of the respective drug than did the tetracycline drugs.

Erythromycin stearate was chosen for oral medication of the breeders based on the greater activity of this drug *in ovo* and in a feed-medication trial with PPLO-infected birds. The latter experiment indicated that erythromycin stearate was more active in controlling tracheal infections caused by PPLO than was oxytetracycline and tetracycline.

Under field conditions, it would be more practical to medicate breeder birds by oral administration. Our experience with commercially available antibiotics indicates that the tetracycline drugs at levels ranging from 250 to 500 Gm. per ton of feed, or equivalent levels in water, failed to control tracheal infections although there was a definite improvement of condition of the birds and greater difficulty was experienced in recovering the organisms from the affected tracheas. This may be due to the poor absorption of the tetracycline drugs from the intestinal tract as reported by Smith.[10] Nevertheless, further studies are necessary to determine whether oral administration of the various drugs in feed or water will prevent egg transmission even though the respiratory phase of the disease may or may not be controlled.

SUMMARY

This series of experiments demonstrated that pleuropneumonia-like organisms, associated with chronic respiratory disease, were transmitted from infected parents to the chicks through the eggs.

Infectious bronchitis virus enhanced the spread and aggravated the severity of latent chronic respiratory disease.

Egg transmission of pleuropneumonia-like organisms to the chicks from infected dams was prevented in two breeding flocks in separate hatcheries by medication of the parent stock.

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Canine Virus Diseases, A Symposium

A forum on canine distemper and canine hepatitis is reported by the *Veterinary Record* (Oct. 15, 1955: 781-790). Participants were: Drs. N. M. Larin, W. Mansi, and R. F. Montgomerie, and Mr. C. E. Woodrow, all of Britain, and Capt. J. A. McBee, U. S. Air Force Veterinary Service. Some of their conclusions follow:

It is impossible to say how much the course of the disease would be altered if a dog in the incubative stage of canine distemper were injected with egg-adapted canine distemper vaccine; the consensus was that the course would not be altered. It showed no benefit as a therapeutic agent.

As to the relative value of egg-adapted vaccine vs. killed vaccine for canine distemper, the opinions differed. The former was preferred when the vaccine was fresh and properly handled, but if not stored under suitable temperature or if adulterated, as with a trace of antiseptic in the syringe, it may be impotent. Furthermore, there appears to be a variation in "batches." Revaccination was recommended, particularly in young dogs. As with other viruses, little is known about this virus and the duration of the immunity. Dogs thought to be immune later came down with some type of infection which, upon ferret inoculation, proved to be canine distemper. The inability of the laboratory workers to reproduce the disease as it is seen in the field remains a puzzling stumbling block to distemper investigation.

The killed vaccine produces an immunity of only limited duration; however, the range of this immunity may be wider than with the egg-adapted vaccine.

When encephalitis follows distemper, some dogs recover but the majority do not. Treatment seems not to be of benefit.

Canine hepatitis is not a disease of young pups. Affected dogs are usually over 3 months of age; younger dogs do not develop the disease even when inoculated.

In America, the hepatitis virus has been found in urine of apparently recovered dogs as long as 272 days after onset of the disease. In England, it had not been found in the bodies of dogs more than 41 days after the primary infection. At one laboratory, the disease could not be transmitted after 15 days from the primary attack.

Dogs which have recovered from hepatitis should not be sold or taken to shows until they have been isolated for at least 30 days after apparent recovery.

The virus has been shown to live in a "freeze-dry" state, in sealed bottles, for five years; it then caused fatal hepatitis in 3 pups.The virus also survived heating at 56 c. for 30 minutes.

However, after a kennel has been infected, it is difficult to tell when the virus has all been destroyed and when dogs taken from this kennel will no longer be carriers.

Wire Encircling Heart of a Normal Steer

With the current interest in reports of heart conditions, the following incident may belong in the records.

At a small country packinghouse, in 1943, a carload of 3-year-old steers was on the floor. While the butcher was preparing to remove the heart from a normal steer, his knife hit metal. On closer examination, the following was observed:

A common galvanized piece of steel wire half the size of iron baling wire, with hooks on either end more complete than a shoe button hook, was found almost completely circling the heart just below its largest muscular circumference, with the hooks pointing outward and perforating the pericardium. All the wire except the hook ends, which were about an inch apart, was inside the heart sac. The perforations made by the ends evidently furnished drainage. No tissue damage or irritation was evident. A slight groove remained in the heart muscle when the wire was removed. The apparently perfect drainage maintained by the perforating wire suggests that such a perforation might be simple to install surgically, should a reason exist.—*V. C. Pauhlman, D.V.M., Prosser, Wash.*

Botulism in Cattle

Six of 7 cows in a stable showed illness, with cessation of rumination and constipation but no fever. Two became paralyzed and died in a few days, others recovered after receiving toxoid. *Clostridium botulinum*, type C, was isolated from the carcass of a cat in the hayloft above the stalls occupied by the cows.—*Vet. Bull., Oct., 1955.*

A Resume on Swine Erysipelas

Swine erysipelas, unlike hog cholera, is not an eradicable disease. The etiological agent seems ubiquitous, some strains apparently having a habitat in the sea as well as in many lands. It is found as a saprophyte on fish and as a pathogen in sea mammals (seals and porpoises); as a saprophyte as well as a pathogen in swine; and at least as a pathogen in several other species of land mammals and birds. It also exists for long periods in the soil, in decomposing organic material, and many other places. Therefore, we must be prepared to live with, and control, the disease.

Evidence of swine erysipelas on this continent was first reported in 1921 and the first epizootics in 1927. Retrospection suggests, however, that the disease probably has been present much longer.

The tales of the old-timers about hogs recovering from cholera, then sloughing patches of skin, and their fixed opinion that feeding new corn in the fall often generated cholera, strongly suggest confusion with erysipelas. Even in this more enlightened period, we know how easy it is to confuse erysipelas with several other diseases.

The fact that erysipelas responds remarkably well, both prophylactically and therapeutically, to biological agents specific for other diseases, particularly anti-hog cholera serum, also indicates how diagnoses which were incorrect could have been erroneously "confirmed" by a "therapeutic diagnosis."

Swine erysipelas has been, for about 70 years, an important disease in Europe, especially in Germany. Unfortunately, a war intervened about the time erysipelas was first intensively studied in this country, so we were denied access to much information gleaned by leading investigators.

Not until about 1939 did we begin to use the simultaneous serum and culture vaccination introduced in Europe by Lorenz about 45 years earlier. And not until about 1949 did we learn how to consistently induce the disease, thus to challenge immunities and make progress with research, by adapting the skin-scarification inoculation method introduced by Fortner about five years earlier. Until recently, most of what we knew about swine erysipelas had been learned the hard way, by practitioners experimenting and observing while battling the disease in the field.

Research Is Now Progressing

Research on erysipelas on this continent is now making commendable progress. In this issue, on pages 277 and 283, are reports which merit study, particularly the clinical phases. If there are variations from one's field observations, they could merely represent the recognized variability of the disease, but they also could be the result of fewer complications under controlled conditions.

Also meriting restudy is the summary of extensive research in Germany by Wellman *et al.* (*see* p. 331 of the October, 1955, JOURNAL). Such articles should be of aid in comprehending how the peculiarities of this variable disease develop.

Also of great interest are abstracts of recent German articles, one on control of erysipelas by Schmidt (*see* p. 281) and one on treatment by Muller (p. 282). A study of the above, along with the several recent reports of research in America on various vaccines, suggests many questions.

Regarding classification, erysipelas is recognized in extreme forms, from peracute to chronic. Using a special classification for animals which have "diamond skin" or "urticarial" lesions would seem to have little justification. The urticarial wheal is but one of the lesions of the acute phase, and the occasional sloughed skin sequela is but a lesion of the chronic phase.

Wellman believes that these wheals quite constantly accompany septicemia. However, if this is true, either it is incorrect to assume that all acute cases are septicemic or the urticarial phase must be so fleeting that it usually is not present at the time the sick pig is examined; it probably would not have time to develop before death in peracute cases. Since these wheals (not readily seen in colored animals) are the only truly pathognomonic lesion of acute erysipelas, they are always carefully searched for in making a diagnosis. Nevertheless, the average estimate by a group of experienced diagnosticians from the "erysipelas belt" was that this lesion has been observed in only 6 per cent of acute cases.

PROPHYLAXIS

Schmidt's report covering a 16-year period in Germany is interesting. Vaccinational erysipelas in only 1 pig in 33,000 is not surprising. It apparently is noticed less often here. Immunity failure in only 1 of 862 pigs when a smaller culture dose was used, and 1 of 2,700 pigs when the dose for all pigs was 1 cc., is indeed satisfactory. If under our present simultaneous vaccination methods we were to have only one "immunity break" in 2,700 pigs, it would suggest that the animals had not been severely exposed. However, if not less than 1 cc. of culture were used here, followed by a "booster" inoculation, as they used it, our records could be as good.

I (W.A.A.) used "booster" doses, without serum, ten weeks after the simultaneous vaccination of baby pigs from 1944 to 1951, with 100 per cent success but not in enough herds to draw conclusions. With a disease as unpredictable as erysipelas, conclusions, on its course in the field, are justified only after years of extensive observation.

The attempt to produce immunity in Germany with a premixed serum and culture certainly indicates a pioneering spirit. Schmidt's reference to vaccinational abscesses following the use of a bacterin is one of the first seen on this seemingly common "side effect."

TRANSMISSION BY VACCINATION

Perhaps the most important of Schmidt's comments is on the improbability of spreading swine erysipelas by using standardized culture. His view is supported on this continent by the many thousands of times that only part of a herd has been vaccinated, with no evidence of spread to the balance of the herd. This vaccination of a fraction of a herd has usually been done unwittingly, when nursing pigs were vaccinated without realizing that their dams seldom if ever retained immunity from similar vaccination a year previously. The frequent references to producing active infection by using vaccination culture have not yet been supported by research. On the other hand, the work of Sikes *et al.* amply demonstrates the ease with which the disease is spread to susceptible contact pigs when more potent organisms are used for inoculations, one of their most significant findings. (Two unvaccinated pigs and 3 which had apparently lost their immunity developed natural infection by contact with pigs infected intravenously.)

Because of evidence that the organism could survive and even multiply in the soil, erysipelas has long been considered a soil-borne infection. Its contagiousness was questioned because of the innumerable times that only one or a few pigs in an untreated herd developed infection. Also, sows have died of acute erysipelas while their untreated nursing pigs remained normal.

A STRESS FACTOR

Such occurrences caused speculation, both in America and in Germany, that a third (stress) factor, in addition to a virulent organism and a susceptible pig, was required to incite infection. Practical observations often indicated that eating an uninfected carcass or gorging on tankage or new corn could "trigger" the infection in a herd. The effect of extremely hot weather has also been suspected. These observations supported the soil-borne and latent infection theories. On the other hand, contact pigs and neighboring herds often sicken in sequence as if the disease were contagious.

Sikes indicates that success in inducing the disease was due, at least in part, to the use of 10 per cent horse serum in the medium for challenge organisms. Could a protein agent likewise activate the organisms in the tonsils and Peyer's patches of "carrier" animals?

More research on the mode of natural infection is sorely needed.

THERAPY

Muller's report endorses American findings on the remarkable therapeutic value of penicillin in acute erysipelas. He suggests that the relapses, following a single dose of penicillin, would be prevented by giving serum also. During severe epizootics in 1948 to 1950, we frequently saw relapses, despite heavy doses of anti-swine erysipelas serum, but the animals again responded when penicillin was repeated. If herds with acute erysipelas are checked daily and penicillin therapy is repeated for those which recover slowly, chronic sequelae can be reduced to nearly zero.

As for the best method of prophylaxis, we must await developments. Recent research indicates that both avirulent vaccines and bacterins have merit. We hope at least one of them will prove satisfactory.

Louis A. Merillat—1868-1956

Dr. Louis A. Merillat, internationally known veterinary surgeon, teacher, journalist, and militant leader of his profession for many years, died on Feb. 25, 1956, at the age of 87. Death came quietly as he sat, reading, at his desk in his home on Chicago's south side where he had lived for many years.

So many honors came to this man, who devoted his life and energies for nearly 60 years to the profession and science he loved, that it is difficult to find new thoughts or words by which to express appreciation of his work and pay tribute to his accomplishments. It can be said that, of the whole picture of veterinary medicine and the profession in America over a span of 68 years, "most of this he saw, and part of it he was."

Add to this markedly active participation in civil professional affairs his active role in veterinary military work in the Army Veterinary Corps in World War I —particularly with the A.E.F. in France— and one gains some idea of the vigorous, often strenuous, life of Louis Merillat as a veterinarian and a patriotic citizen.

Since the true measure of a man is in the record he leaves of his life and work, it is well to cite again the highlights of a career that brought recognition and honors to one who lived an unusually full life.

Born in Wooster, Ohio, March 22, 1868, the son of David and Cecil (Guerne) Merillat received his early education in the local public schools and graduated from Mt. Eaton high school in 1884. He then entered Ontario Veterinary College, affiliated with the University of Toronto, graduating in 1888 with the twenty-third class to come out of that school.

Dr. Merillat came to Chicago and started practice in 1888. Three years later he married Mary Smith; they had one son, Louis, who graduated from West Point Military Academy in 1915, and went on to a distinguished career in the Army.

Shortly after starting his practice, Dr. Merillat joined the faculty of McKillip Veterinary College, where he did a great deal of equine dentistry, this experience leading to the publication of his first article in a veterinary journal (1898) and, eventually, to his first book, "Animal Dentistry," in 1903. In addition to his teaching work, Dr. Merillat served as secretary at McKillip from 1892 to 1900.

Dr. Louis A. Merillat

Leaving McKillip in 1901, Dr. Merillat accepted a position as professor of surgery of the Chicago Veterinary College where he served until 1916; he also became associated with Dr. J. M. Wright in a busy veterinary hospital and practice, a partnership that continued until 1917, when Merillat was commissioned in the Army Veterinary Corps and left Chicago to start his two and one-half years of military service.

Commissioned as major in the Veterinary Corps, U. S. Army, in October of 1917, Dr. Merillat served first with the 41st Division at Camp Greene, N. Car., and became division veterinarian when, later that year, the 41st was moved to Camp Mills, N. Y., for shipment overseas as part of the A.E.F. In January, 1918, he was made commanding officer of the advanced section veterinary hospital at Neufchateau (Vosges), France, and continued in that capacity until appointed in March as executive officer of the Franco-American Veterinary Liaison Mission in Paris, an assignment he carried out with characteristic ability and distinction. In August, 1918, he was made chief veterinarian of the First Army, A.E.F., and so served until February, 1919.

For his outstanding services in collaboration with French Army veterinarians, an assignment for which he was especially fitted, Colonel Merillat was made a Cheva-

lier of the French Legion of Honor in 1919.

Upon returning to civil life, Dr. and Mrs. Merillat went back to the old homestead in Ohio for a time and then returned to Chicago where he was active in veterinary publications work for the balance of his career. He was an associate editor of *Veterinary Medicine* from 1913 to 1921 and its managing editor in 1921-1923. From 1924 to 1929, he, with Dr. J. V. Lacroix, edited *North American Veterinarian*. At one time, he also edited the *Haver-Glover Messenger*.

Dr. Merillat's first connections with AVMA affairs were in 1906-1907 when he was elected as vice-president, and in 1916-1917 when he served as secretary before entering military service. He was elected president of the Association in 1924-1925 at the Des Moines meeting. From 1928 to 1938, he was Executive Board member from District III. When the reorganization of the AVMA headquarters work was undertaken in 1939, he again served as executive secretary for two years, 1939-1940, and as editor-in-chief of the Association's publications from 1940 to 1950 inclusive. He retired in 1950 from active direction of the publications work and was editor emeritus until his death.

In 1943, he was made an Honor Roll member of the AVMA, following 50 years of active membership.

In 1945, he received the Twelfth International Veterinary Congress Prize, conferred by the AVMA for "outstanding service to the veterinary profession."

In 1948, when he celebrated his sixtieth year in the veterinary profession, Dr. Merillat returned to his alma mater, Ontario Veterinary College, where he was honored for his work as "teacher, author, and champion of the veterinary profession."

In 1950, he was the second recipient of the AVMA Award in recognition of his "57 years of service to the Association as member, executive secretary, president, editor, and always inspiring leader."

As an author, Louis A. Merillat is known for the numerous articles published in veterinary journals over the years; he was a prolific writer on the whole gamut of veterinary scientific and professional subjects. In the early days, he authored five texts: "Animal Dentistry" (1901); "Principles of Veterinary Surgery" (1906); "Veterinary Surgical Operations (1907); "Treatment of Wounds" (1913); "Treatment of Colics of the Horse" (1914).

His work (with the late D. M. Campbell) in writing and producing the two-volume "Veterinary Military History" (1935) was an ambitious undertaking which few men would have shouldered, but it gave him an opportunity to sum up the early history of veterinary medicine and of veterinary services, subjects he never tired of, and to express his viewpoints on veterinary medical affairs and personalities in this country, subjects on which he never hesitated to speak forthrightly, whether or not his colleagues agreed with him.

Dr. Merillat liked to be regarded as a "radical" in veterinary medical affairs. He had little regard for the "Tory." He once said, "The radical makes errors because he takes chances, but [he] makes the double plays and bats in the runs. The Tory makes only errors of omission and stays in the minors. He's the stand-patter of veterinary progress of whom there are not a few."

"L. A." never stood pat; he wound up his memoirs (which were published in the JOURNAL several years ago) from which the above quotation is taken by saying, "These sketches have been intentionally critical rather than complimentary toward men and the things men do. If anyone was hurt, there is solace in [knowing that] compliments are not legal tender. Moreover, biographers seldom get to work 'til the 'guy' is dead."

Dr. Merillat is dead. He will be sorely missed by all who knew and loved him, for he had an infinite capacity to make friends; many persons in the profession disagreed at times with his viewpoints but respected his forthright expression of them. To do justice to Dr. Merillat's life and work, a book should be written; here are only a few highlights.

Surviving are two granddaughters, both of Chicago: Mrs. Christopher Choporis who, with her husband and son, had lived with and cared for Dr. Merillat in recent years, and Miss Ethelwynne Merillat.

Memorial services for relatives and friends were held in Chicago on February 29. These were followed on March 1 by services in the chapel at Fort Myer, with interment in Arlington National Cemetery at which full military honors were accorded the deceased.

Current Literature

FOREIGN ABSTRACTS

Antihistamine in Bovine Indigestion

This paper is largely a review of the literature on the effect of histamines on digestive disturbances of cattle. Atony of the rumen can develop when excessive amounts of histamine are released as a result of an alkaline pH, or feeding excessive amounts of proteins. Experimentally, use of histamines can also produce paralysis of the rumen musculature. This condition can be prevented or counteracted by the use of antihistamines. The use of neo-antergen® (Merck) is reported in clinical cases. Good results followed the intramuscular injections of 20 cc. every 12 hours. The author believes that the complete pharmacodynamics of antihistamines remain to be determined.—[*R. Florio: Les antihistaminiques de synthèsesdans le traitement des indigestions, chez les bovins. Rev. méd. vét. (Dec., 1955): 766-773.*]—R.F.V.

Extrauterine Pregnancy in a Ewe

The author states that extrauterine pregnancy is not rare in women but apparently is not observed frequently in animals. A review of the literature by Romani records one abdominal pregnancy in ewes and the author reports another. The ewe had been bred for approximately seven months and died 14 days after developing inappatence, loss of condition, and general debility. Necropsy revealed a fully developed fetus with signs of resorption attached to the walls of the omentum and rumen. The reproductive organs appeared to be normal with no scars of the uterus to indicate the possibility of a rupture. Death was attributed to peritonitis.

The author states that at no time were there any indications that the ewe was pregnant; the vulva appeared normal, and there was no development of the mammary glands. He concluded that fertilization occurred outside the uterus in this case.—[*M. Berthelon: Observation de gestation extra-utérine primitive, chez la brevis. Rev. méd. vét. (Dec., 1955):721-723.*]—R.F.V.

Response of Cows to Sun Radiation

Respiration rate and rectal temperature rose sharply when cows were exposed to the sun during the late morning. Cows in the shade during this time were affected to a much lesser degree.

Drinking cool water caused a drop in rectal temperature but did not affect the respiration rate markedly when a cow was moved to shade from its exposed position. After one hour in the shade, the cool water drink lowered both rectal temperature and respiration rate.

A cow which drank cool water one half hour before exposure to the sun did not show a rectal temperature rise for a half hour after being in the sun. The respiration rate did go up.

It appears to the author that drinking cool water aids in the regulation of body temperature, but seems to have no significant effect on the respiration rate. He emphasizes that cattle should be provided with shade and fresh, cool drinking water.—[*A. Berman and R. Volcani: Response of Cows to Different Intensity of Sun Radiation. Refuah Vet., 12, (June, 1955): 203, 235.*]—M.E.

Ocular Vaccination Against Newcastle Disease

The Beaudette vaccine strain of Newcastle disease (ND) virus, having a hemagglutination titer of 1:2,048 to 1:4,096 and previously used intramuscularly was used for this vaccine.

The vaccine is prepared by adding nine parts of glycerine containing 1:8,000 of crystal violet solution to one part of ND allantoic fluid virus. A dose of 0.05 cc. is placed in the eye of 21-day-old chicks.

Eight to 10 vaccinated chicks were challenged after three, five, seven, and nine months. All challenged chicks were resistant, all controls died. —[*Cordier-Boullangier, A. Ounais, and B. Harouni: Ocular Vaccination Against Newcastle Disease. Rec. Méd. Vét., Alfort, 131, (1955): 765-761.*]—J.P.S.

Parasitic and Nonparasitic Eosinophilia

A comparative study was made of hypereosinophilia (parasitic vs. nonparasitic) in man, cats, dogs, and guinea pigs. That induced by parasites may be differentiated from others, using the three following criteria: (*a*) frequency of response to a given agent; (*b*) shape of the curve of eosinophilia; and (*c*) instability, as contrasted to irreducibility, in patients treated with hormones.

Among parasitic cases; hypereosinophilia is almost evident, although varying in degree. Evolution follows the same general curve with lag period, increase, and decrease. Eosinophilia is generally irreducible in patients treated with hormones.

Nonparasitic cases are much more diversified but usually show: (*a*) low frequency of response (streptomycin or red pepper cause eosinophilia in 10 to 40 per cent of treated patients); (*b*) a flattened and irregular curve (with exception for tropical or radium-induced eosinophilia); (*c*) easy remission following treatment with corticotropic hormone or with cortisone, again with the exception of eosinophilia induced by x rays or radium.

Response to hormonal treatment depends on the severity of intoxication by the causal agent. In the guinea pig, experimental intoxication by extracts from *Fasciola hepatica* induces congestive and degenerative changes in the glandular cells of the adrenal glands, whereas after injection of streptomycin, or of red pepper, histopathological responses are limited to juxtamedullary congestion and hyperplasia in the reticulated area.

The hematological syndrome of eosinophilia thus appears to be correlated with ailments of the adrenal glands. (Author's summary).—[*R. Deschiens: A Study of Parasitic and Non-Parasitic Eosinophilia. Rev. Path. Gén. et Comp., Paris (1955): 1316-1335.*]—J.P.S.

BOOKS AND REPORTS

Diseases of Goats

This publication is the first complete textbook dealing with the diseases of goats. It has been written in several chapters, each of which is devoted to an organic system of the goat, discussing its anatomy, physiology, husbandry, and pathological disorders.

The first chapter deals with digestive organs, the second with reproductive organs. The third chapter describes normal and pathologocial conditions of the udder, with further chapters devoted to the diseases of the skin, respiratory, circulatory, and urinary systems.

The last part of the publication discusses infectious diseases of goats.

This interesting publication has been written clearly and the many illustrations are helpful. The practitioners who deal with goats will find this book useful.—[*Disease of Goats. (Die Krankheiten der Ziege). By A. Honeker. 529 pages. 202 illustrations. H. Wellershaus, Dortmund, Germany. 1950. Price not given.*]—F. KRAL

Diseases of Pigs

This fourth edition of a British text first published in 1940 has 15 chapters with the last ten devoted to diseases of swine, disease-producing agents, and diagnosis.

The first chapters present concise descriptions of 52 modern breeds of swine; breeding and market types; housing and management; feeding; and anatomy, health, and handling of swine.

The text covers the field of swine problems quite completely but in a brief and general way. It is written chiefly for British use so is better adapted for their swine problems than for those in America.

Exception can be taken to a few statements such as: . . . boars not reaching "maturity for breeding" until 18 months of age (many are in service at 8 months); that swine fever (cholera) can be deterred by "avoidance of crowding" or "proper feeding and breeding"; and the implication that "purulent conditions—with abscess formation" are necessary in castration wounds for the development of tetanus.

It is a well-written, thought-provoking text and well worth reading. The discussion of respiratory diseases is particularly good.—[*Diseases of the Pig. By David J. Anthony. 4th ed. 369 pages. Illustrated. Williams and Wilkins Co., Baltimore, Md. 1955. Price not given.*]—W.A.A.

Cesarean Section in Swine and Ruminants

This publication deals with operative obstetrics in swine and cattle. It discusses briefly the indications for cesarean section, and the necessary arrangements which should be made before the operation. For local anesthesia, 2 per cent solution of novocain or isocain is applied to the skin, subcutis, muscles, and peritoneum.

For cattle, the paravertebral anesthesia is recommended. Eqidural anesthesia is not sufficient for a cesarean section.

For general anesthesia in swine, intravenous injections of chloral hydrate, eunarcon, or nembutal® are recommended.

In further chapters, the technique of the cesarean operation in swine and cattle is described.

A special chapter deals with different pathological conditions which should be considered as a risk with regard to the anesthesia and the operation itself.

The final chapter discusses the feeding of animals after the operation, particularly when infection with *agalactiae* develops, and the artificial feeding of the young.

The publication has some good illustrations and may serve as a help for practitioners.—[*Cesarean Section in Swine and Ruminants. By H. Tillman. 102 pages. 38 illustrations. Paul Parey, Berlin, Germany. 1954. Price 8,60 marks.*]—F. KRAL.

Textbook of Embryology of Domestic Animals

Two well-known experts, veterinary anatomist Zietzschmann and veterinary embryologist Krolling, present in this textbook an extensive work on the embryology of domestic animals. The material has been arranged in a clear and logical sequence. It deals first with the development of male and female cells, and describes the sexual cycle and fertilization. Further chapters describe the development of the embryo, beginning with blastogenesis and continuing through the further stages of the developing fetus.

The development of the skin and adnexa, including the hoofs, claws, and nails, followed by description of the development of the nervous, circulatory, musculoskeletal, genitourinary, digestive and respiratory systems, are further chapters.

This very important fundamental work in veterinary medicine has been written clearly in all details. The numerous illustrations are easily understood and helpfully supplement the text. It may serve not only students of veterinary medicine but also as a good source of information for practitioners.—[*Textbook of Embryology of Domestic Animals. By O. Zietzschmann and O. Krolling. 482 pages. 597 illustrations. 2nd ed. Paul Parey, Berlin, Germany. 1955. Price 88 marks.*]—F. KRAL.

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Dr. Peterson Appointed Acting Dean at Oklahoma Veterinary School

Dr. Duane R. Peterson (KSC '45) was appointed, on Jan. 28, 1956, as acting dean of the School of Veterinary Medicine at Oklahoma A. & M. College, Stillwater, to fill the vacancy left by the recent death of Dr. H. W. Orr. Dr. Peterson has been head of the Department of Anatomy of the School of Veterinary Medicine for several years.

s/C. H. McElroy.

AMONG THE STATES AND PROVINCES

California

Tulare County Veterinarians.—The Tulare County Veterinarians elected the following officers at a dinner meeting in Dinuba on Jan. 12, 1956: Robert Dove, Porterville, president; Carl T. Lambert, Visalia, vice-president; and R. B. Barsaleau, Visalia, re-elected secretary-treasurer. This group has been active in bringing data related to the new brucellosis control program to the meetings for discussion and clarification when needed. Also, the Tulare County Veterinarians have been holding the rabies vaccination clinics (in cooperation with the local public health department) on a pooled vaccine basis and thus professional income from time spent at the clinics is equal among the participants.

s/R. B. Barsaleau, *Secretary.*

Colorado

Dr. Aichelman Appointed to Health Board.—Dr. W. W. Aichelman (COL '32) Fort Lupton, has been appointed as a member of the Colorado State Board of Health to fill the vacancy created when Dr. Paul Pattridge, formerly of Golden, moved to Batesville, Miss.

Dr. Aichelman has been a member of the Colorado Veterinary Medical Association for 25 years. He served on the Fort Lupton City Council for ten years, six years as mayor.

s/G. H. Gilbert, *Secretary, Colorado V.M.A.*

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Dr. Haas Writes from Hawaii.—Dr. Walter Haas, past secretary of the Colorado Veterinary Medical Association, writes from the beach at Waikiki that he and his family are preparing to move to the Island of Maui where Dr. Haas will assume regulatory duties as a territorial veterinarian. Included in his jurisdiction will be Kalawao County, the leper colony on the Island of Molokai.

s/G. H. Gilbert, *Secretary, Colorado V.M.A.*

Connecticut

Fairfield County Association Officers.—At the regular annual meeting of the Fairfield County Veterinary Medical Association, held at Cobbs Mill Inn, Westport, on Dec. 14, 1955, the following officers were elected: Maurice H. Ryan, Stamford, president; George Pickett, Stratford, president-elect; and William R. Leggett, Westport, secretary.

s/William R. Leggett, *Secretary.*

Indiana

Officers of State Association.—The seventy-second annual convention of the Indiana Veterinary Medical Association, Jan. 11-13, 1956, was one of the most interesting and best attended (492) to be held by this group. At this meeting, the following officers were elected: J. E. Jordan, Indianapolis, president; R. W. Worley, South Bend, president-elect; R. F. Portman, Lafayette, vice-president; and L. M. Borst, Indianapolis, re-elected secretary-treasurer. The new members of the board of directors are Galen E. Krill, Boswell, representing the Northwestern Indiana District V.M.A.; B. V. Allen, Fort Wayne, the Northeastern District; Thomas W. Freas, Veedersburg, the Sixth District; and Odell Archer, Terre Haute, the Indiana-Illinois District.

s/J. L. Kixmiller, *Resident Secretary.*

Maryland

Techniques for Tuberculin Testing.—On Nov. 19, 1955, 15 regulatory and practicing veterinarians from the eastern shore of Maryland visited the Maryland Livestock Sanitary Service Laboratory at Salisbury to witness a demonstration of proper techniques for injecting tuberculin and for interpreting reactions to the tuberculin test in cattle.

Dr. M. H. Trout, veterinary inspector for the Maryland State Board of Agriculture at Salisbury, who is the regulatory official for the eradication of tuberculosis and brucellosis in the area, demonstrated the injections and readings in the cattle.

Injections were made into the caudal fold and vulva of several animals that had previously been sensitized with a killed culture of the tuberculosis organism. These animals exhibited typical reactions at both sites. Several negative animals were also available for comparison.

Dr. I. M. Moulthrop, in charge of the Branch Laboratory of the Maryland Livestock Sanitary Service, arranged the demonstration since more and more "straight kill' animals are being found in slaughterhouses with lesions of tuberculosis. Many of these animals have originated in herds tested annually without reactors.

Although there have been no reports of such animals from the eastern shore of Maryland, it is hoped that this demonstration will be helpful in the prevention of such cases.

Michigan

The Thumb Association.—The Thumb Veterinary Medical Association met in Caro on January 12 to honor members of the association who had served their communities for 40 years or more. Dean C. F. Clark, of the College of Veterinary Medicine, Michigan State University, presented the following men with plaques commemorating their achievements: John McCoy (ONT '01), Minden City; Frank Harrison (ONT '07), Bad Axe; Thomas McAlister (ONT '11), Kinde; Peter Keyser (GR '11), Port Huron; M. J. Geiger (CVC '12), Croswell; A. T. McIntyre (CVC '12), Brown City; Henry Jurgess (GR '13), Harbor Beach; J. W. Harrison (MCK '14), Sandusky; Ray Baraclow (GR '14), Fairgrove; and E. J. Reynolds (GR '14), Deckerville.

s/R. S. JOHNSON, *Secretary.*

Missouri

Greater St. Louis Association.—At the February 3 meeting of the Greater St. Louis Veterinary Medical Association, in the Sheraton Hotel, Dr. Mina Edelman, radiologist, discussed "Radiology for Veterinarians."

s/ALLEN B. SHOPMAKER, *Secretary.*

Montana

Dr. Safford Appointed Assistant State Veterinarian.—Dr. J. W. Safford, who has been a member of the faculty of the Montana State College for the past four years, recently resigned to accept a position as assistant state veterinarian with the Livestock Sanitary Board in Helena. His successor, Dr. J. E. Catlin (GA '54), in addition to teaching will have charge of the college and experiment station livestock and will be co-leader on the research project "Urolithiasis of Range Cattle."

s/E. A. TUNNICLIFF,
Montana State College.

North Dakota

Short Course.—The North Dakota veterinary short course was held at the Van Es Laboratory, Fargo, on Feb. 13-14, 1956.

The program listed the following speakers and their subjects: E. A. White (Tips to Practitioners, and Acetonemia and Milk Fever); H. L. Easterbrooks, Storrs, Conn. (Sterility and Abortions, and Vibriosis); P. O. Triggs, M.D. (Antibiotic Sensitivity); M. L. Buchanan and F. M. Bolin, Fargo (Dwarfism in Cattle); G. Christensen, Minot (Beef Cattle Problems); I. A. Schipper and W. E. Dinusson, Fargo (Parakeratosis of Swine); S. H. Richards, Fargo (Lymphocytic Choriomeningitis); Carl Norden, Jr., Lincoln, Neb. (Swine Diseases, and Cholera-like Diseases); and R. F. Shumard, Fargo (Trends in Parasite Control).

Members of the staff discussed and demonstrated some animal disease problems.

Oklahoma

Oklahoma County V.M.A. Officers.—The following officers have been elected to serve the Oklahoma County Veterinary Medical Association for 1956: W. K. Fauks, Oklahoma City, president; and James M. Brown, also of Oklahoma City, secretary-treasurer.

s/JAMES M. BROWN, *Secretary.*

Pennsylvania

Dean Allam Visits Veterinary School in Mexico.—Dean Mark W. Allam of the School of Veterinary Medicine, University of Pennsylvania, in January visited the National School of Veterinary Medicine at Mexico City, Mex. He served as a consultant to the faculty there under the auspices of the Pan American Sanitary Bureau, World Health Organization.

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Dr. Floyd Cross, Fort Collins, Colo., president of the AVMA, and Dr. Norman Topping, vice-president in charge of medical affairs of the University of Pennsylvania, at the annual conference for veterinarians Jan. 3-4, 1956.

Tennessee

Officers of State Association.—At the annual meeting of the Tennessee Veterinary Medical Association in Nashville, Jan. 15-17, 1956, the following officers were elected: H. W. Nance, Lawrenceburg, president; Hugh L. Lamb, Athens, president-elect; E. M. Spence, Jackson, vice-president; and H. W. Hayes, Knoxville, secretary-treasurer. The following men were elected to the board: H. L. Johns, Huntingdon; H. H. Morgan, Clarksville; and W. C. Cook, Knoxville.

s/HUGH L. LAMB, *Resident Secretary.*

Washington

Conference for Veterinarians.—The eighth annual Washington state conference for veterinarians will be held at the College of Veterinary Medicine, Washington State College in Pullman, April 2-4, 1956.

In addition to members of the staff, the following authorities will present papers at the meeting: Floyd Cross, Fort Collins, Colo., president of the AVMA (Sheep Diseases); R. M. Cello, Davis, Calif. (Feline Diseases); Jacques Jenny, Philadelphia (Canine Surgery); A. H. Quin, Kansas City, Mo. (Swine Diseases); F. G. Fielder, Bloomington, N. J. (Radiology); F. A. Todd, Washington, D. C. (Exotic Diseases); G. T. Edds, Fort Dodge, Iowa (Anesthesia); and D. V. Zander, Kirkland, Wash. (Poultry Diseases).

A feature of the conference will be a dairy cattle disease forum conducted by northwest veterinarians, with Dr. Ab Quin as moderator.

The session will again be highlighted by a closed-circuit television of surgical and other procedures.

A registration of 250 veterinarians and 100 women's auxiliary members is expected.

s JOHN R. GORHAM, *Conference Chairman.*

Wisconsin

Officers of Milwaukee Association.—At a recent meeting of the Milwaukee Veterinary Medical Association, the following men were elected: Charles J. Gurneau, Racine, president; George F. Lynch, Milwaukee, vice-president; and John M. Wenzler, Milwaukee, secretary-treasurer.

s/JOHN M. WENZLER, *Secretary.*

STATE BOARD EXAMINATIONS

Texas—The Texas State Board of Veterinary Medical Examiners announces that the next veterinary licensing examination will be held on May 28-30, 1956, at Texas A. & M. College, College Station, Texas. The completed applications must be returned to the following address not later than 30 days before the examination date. Requests for applications and additional information should be addressed to: Mr. T. D. Weaver, executive secretary, Texas State Board of Veterinary Medical Examiners, 520 Littlefield Bldg. Austin 15, Texas.

North Carolina—The North Carolina Veterinary Medical Examining Board will meet at Greensboro, N. Car., on June 25, 1956. James I. Cornwell, 65 Beverly Rd., Beverly Hills, Asheville, N. Car., secretary.

DEATHS

★**Adolph Eichhorn** (BUD '95, NYA '00), 82, Pearl River, N. Y., died Jan. 23, 1956, at his winter home in Miami Beach after a short illness.

Noted authority in animal pathology and infectious diseases of animals and one-time chief of the pathological division of the Bureau of Animal Industry, U.S.D.A., Dr. Eichhorn was born in Hungary in 1875, was educated at the Royal Veterinary College, Budapest, 1892-1895,

Dr. Adolph Eichhorn

and received the D.V.S. degree from New York-American Veterinary College in 1900. He served on the faculty there for a year and then entered the meat inspection service of the BAI, where he served for five years. In 1906, he transferred to the pathological division where he remained for ten years, being chief of the division in 1915-1916.

In the latter year, Dr. Eichhorn resigned from the Bureau to become head of the veterinary department of Lederle Laboratories at Pearl River. He returned to the Bureau in 1938 as director of the Animal Disease Station at Beltsville, Md., where he remained until 1943. Since 1943, he has been a consultant to the Lederle Laboratories Division of American Cyanamid.

While in Washington with the Bureau, he was part-time professor of veterinary medicine at George Washington University for two years and later (1917-1921) was on the veterinary faculty at the New York University. He was a prolific writer on a variety of veterinary subjects and is best known in this field as one of the translators and editors, along with the late Dr. John R. Mohler and others, of some editions of Hutyra and Marek's works on diseases of domestic animals.

He was an active member of a number of professional and scientific societies for many

years, including the AVMA which he joined in 1901 and in which he attained honor roll status five years ago.

Dr. Eichhorn is survived by his widow, a son, Dr. Erwin Eichhorn (UP '35) of Rio de Janiero; a brother, and two sisters.

★**Orvin T. Hayer** (CVC '15), 73, Bloomington, Ill., died Dec. 23, 1955, as the result of an automobile accident. Dr. Hayer had been McLean County veterinarian for 13 years and for many years had assumed the task of testing animals prior to exhibition at the McLean County 4-H fair. He is survived by his widow, three sons, a daughter, nine grandchildren, and two great grandchildren.

Dr. Hayer was admitted to the AVMA in 1933.

Harry B. Hickman (KSC '20), 62, Sweet Springs, Mo., died Nov. 24, 1955. Dr. Hickman had practiced for many years in Malta Bend, Mo., before locating in Sweet Springs. He had been a member of the AVMA.

Troy S. Hopkins (IND '22), 62, Montgomery, Ala., died Dec. 5, 1955. After practicing a year, Dr. Hopkins served with the U. S. Bureau of Animal Industry for more than 30 years. His work in various parts of the country included tick eradication, foot-and-mouth disease, and meat inspection. Dr. Hopkins was a member of the Alabama Veterinary Medical Association.

James W. Hughes (USC '18), 57, Elkton, Md., died Nov. 9, 1955. Dr. Hughes had served in the state senate and also as police magistrate. He retired in 1954 due to failing health. Dr. Hughes had been a member of the AVMA.

Early E. Joiner (TEX '43), 52, Midland, Texas, died Dec. 16, 1955. Dr. Joiner had been a member of the AVMA. He is survived by his widow and a son.

★**Herbert H. Kettler** (OSU '16), 66, Milford Center, Ohio, died Dec. 10, 1955. Dr. Kettler, a general practitioner, was a member of the Ohio State Veterinary Medical Association and of the AVMA.

Albert R. Koen (KCV '06), Los Angeles, Calif., died April 24, 1955. He had been employed by the U. S. Department of Agriculture but had retired in 1939. He had been a member of the AVMA.

★**Louis A. Merillat** (ONT '88), 87, Chicago, Ill., died Feb. 25, 1956. An obituary appears on page 319 of this JOURNAL.

★**William F. Osborn** (KCV '08), 79, Corvallis, Ore., died in 1955 (date not known). Dr. Osborn had been employed by the U. S. Department of Agriculture (BAI) but had retired in recent years. He was a member of the National Association of Federal Veterinarians and of the AVMA which he joined in 1919.

★**Benjamin A. Pomeroy** (MON '83), 94, St. Paul, Minn., died Jan. 12, 1956. Dr. Pomeroy had practiced in St. Paul since 1886, and often spoke of the city's growth from the horse-drawn carriage to modern mechanized transportation. Keeping step with the changing times, his practice changed from one almost exclusively of horses to one of small animals.

Dr. Pomeroy's three sons are also veterinarians. Harold (COL '48) and James (KSC '49) worked with him and Benjamin (ISC '33) is a Professor in the School of Veterinary Medicine, University of Minnesota, in St. Paul. His widow also survives.

Dr. Pomeroy was admitted to the AVMA in 1926.

★**Benjamin H. Priest** (CVC '07), 62, Bakersfield, Calif., died May 17, 1955. Dr. Priest had retired from practice. He is survived by his widow and two children. He was admitted to the AVMA in 1910.

Thomas H. Ruth (CVC '06), 73, Jackson, Minn., died Dec. 16, 1955. Dr. Ruth practiced in DeSmet, except for two terms as state veterinarian, until 1938 when he moved to Jackson, Minn. Illness forced his retirement recently. While in DeSmet, he was active in civic and professional affairs, serving on the board of education, the fire department, and other organizations. Dr. Ruth is survived by his widow and four children.

Edward H. Schirmer (STJ '09), 69, St. Joseph, Mo., died Dec. 29, 1955. Dr. Schirmer had been a poultry inspector with the U. S. Department of Agriculture since 1946. He is survived by his widow and a son.

★**Thomas A. Schneekloth, Sr.** (CVC '02), 77, Lodi, Wis., died Jan. 9, 1956. Dr. Schneekloth had practiced in Lodi for 54 years. He is survived by his widow; a son, Thomas A., Jr. (ISC '38), who practiced with him; one daughter, and four grandchildren.

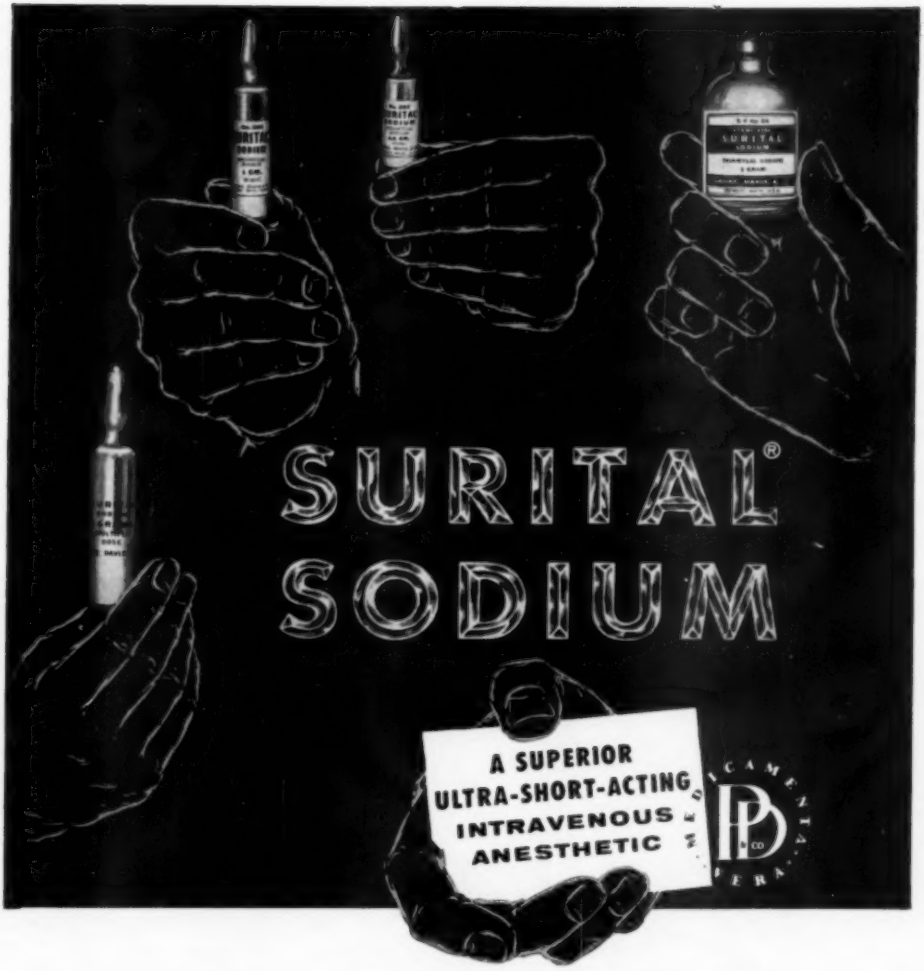
Dr. Schneekloth was admitted to the AVM. in 1942.

Carl A. Schulz (STJ '09), 69, Independence, Mo., died Nov. 25, 1955. Dr. Schulz practiced in Ellsworth, Kan., until 1913 when he opened his practice in Independence. He is survived by his widow, one daughter and two sons, Drs. C. W. Schulz (KSC '34) and N. E. Schulz (Mo '52), both of whom practice in Missouri.

George H. Woolfolk (KCV '06), Brandenberg, Ky., died (date not known). Dr. Woolfolk had retired from service with the U. S. Department of Agriculture (BAI). He had been a member of the AVMA.

John H. Zollinger (UP '02), 76, Brookline, Pa., died Dec. 6, 1955. Dr. Zollinger was for many years a district inspector with the U. S. Bureau of Animal Industry. After his retirement 12 years ago, he was inspector for a meat-packing company. Dr. Zollinger was a member of the Pennsylvania Veterinary Medical Association and had been a member of the AVMA. He is survived by his widow and a daughter.

★Indicates members of the AVMA.



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PROFESSIONAL LITERATURE AVAILABLE ON REQUEST

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ORGANIZATION SECTION

San Antonio Is 1956 AVMA Convention Site

Ninety-Third Annual Meeting Will Be Held October 15-18

Postconvention Tour to Mexico City

For the first time, the AVMA will convene in Texas, in one of the most historic spots in the great Southwest. With its colorful present and romantic past, San Antonio is known far and wide as one of America's truly different cities.

Founded in 1718, the flags of six countries have flown over it—Spain, France, Mexico, the Republic of Texas, the Confederate States of America, and the United States. Site of the Alamo, which has been called the "Cradle of Texas Liberty," San Antonio is also famed for other historic missions, making the city one of the most unusual and interesting in America. With a population of nearly a half-million, it is the third largest city in Texas and one of the nation's fastest growing.

October Meeting Dates

Another unique feature of the 1956 convention arises from the decision to hold it in October, that month having been suggested when the invitation from San Antonio was first considered. This will be the first time in over 35 years that the AVMA has convened later than September and only the third time that it has done so—the 1893 meeting in Chicago was in October and the 1919 meeting in New Orleans was in November.

Postconvention Tour to Mexico City

Plans are under way for an "adjourned final session" in Mexico City on Saturday, October 20, following the close of the sessions in San Antonio

(*Continued on p.* 20)

An aerial view of San Antonio, the AVMA 1956 convention city.

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(*Continued from p.* 18)

the previous Thursday. This adjourned session will be held at the splendid new veterinary school of the National University of Mexico at fabulous, modern University City.

Arrangements approved by the Executive Board have been completed with Happiness Tours, Inc., Chicago, which will offer a selection of tours to Mexico City and other points. Full details will be published in subsequent issues of the JOURNAL and information will also be mailed direct to AVMA members in due time.

Housing Bureau Will Handle Hotel Reservations

In collaboration with the Committee on Local Arrangements, a housing bureau will be set up by the San Antonio convention bureau so that hotel reservations will be handled satisfactorily. The hotel information and reservation form will be published in the JOURNAL in the near future, including information about the excellent motel facilities that will be available for those who drive to San Antonio and prefer such accommodations. All hotels, motels, and public meeting places in San Antonio are completely air-conditioned, as is the Auditorium where most of the convention sessions will be held.

The Alamo, shrine of Texas liberty, in San Antonio.

The Arneson River theater in the heart of the San Antonio downtown area.

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Kodachrome Slides Available

In the Feb. 1, 1956, JOURNAL (adv. p. 18), it was stated that rinderpest slides prepared by the Armed Forces Institute of Pathology, Washington, D.C., were available from the AVMA film library.

This was an error as they must be obtained from the AFIP. Following are the slides available on foreign animal diseases:

To aid in the recognition and diagnosis of foreign animal diseases which are a hazard to the U. S. livestock industry, the Veterinary Registry of the Armed Forces Institute of Pathology has prepared additional visual aids on rinderpest. This material consists of a study set of 76, 2 x 2 kodachrome slides showing gross and microscopic lesions and 25 tissue sections. These sets are accompanied by commentaries for each of the slides and sections and a reprint "Pathology of Rinderpest," by Maurer, Jones, Easterday, and Detray.

The study sets may be used to advantage in connection with the colored sound movie on rinderpest recently prepared by the Federal Civil Defense Administration, Agricultural Research Service, and the Department of Defense, since the studies of the same research team were utilized as the basis for both the study sets and the movie.

A similar study set on African swine fever is now being prepared. In view of the similarities between the lesions of African swine fever and hog cholera, this set will include a comparative set of photographs and sections on hog cholera. A movie on African swine fever in comparison with hog cholera is also in preparation.

These sets may be secured by professional groups or individuals for loan periods of two weeks by letter application to The Director, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, 6825 16th St., N.W., Washington 25, D. C.

There is no charge for the use of the material except for the payment of shipping costs.

WOMEN'S AUXILIARY

President—Mrs. Earl N. Moore, 636 Beall Ave., Wooster, Ohio.

Secretary—Mrs. F. R. Booth, 3920 E. Jackson Blvd., Elkhart, Ind.

Use AVMA Radio Scripts to Further Good Public Relations.—Did you know . . .

A recent survey (*Veterinary Bulletin Lederle*, Summer, 1955) among animal owners showed that (1) Opinions of the veterinary profession were formed entirely from personal experiences with veterinarians; and (2) there is an over-all desire on the part of animal owners to know more about veterinarians and what they do. Three out of four of 874 animal owners said

(*Continued on p. 24*)

Additions to AVMA Film Library

The Keystone of Quality Milk

	Color;		
16 mm.	Running time	Produced by	Rental
Sound	28 min.	Cornell University	$2.50

This film shows how the control of mastitis is dependent on strict sanitation on the farm, early diagnosis and treatment, and the cooperation of the farmer, veterinarian, and state facilities. It is a very good film, with some new ideas on cow barn sanitation. The purpose is to demonstrate a method of combating mastitis, as used in New York State. It shows how the dairy farmer is able to institute mastitis control in conjunction with the local practicing veterinarian, the county agent, and the State Mastitis Diagnostic Laboratory. It also presents some aspects of barn sanitation and milking procedures which are beneficial in the prevention of mastitis.

The photography in this color and sound film is exceptionally good. It is recommended for both professional and lay audiences interested in milk production or the dairy farm.

Friend of a Friend

		Produced for Gaines	
	Black and white;	Dog Research Center	
16 mm.	Running time	by Institute of Visual	Rental
Sound	15 min.	Training, Inc., New York 17, N.Y.	$2.50

This film portrays a veterinarian in small animal practice. The necessary background of veterinary education, research, and postgraduate training is cleverly woven into the story intended to depict an average day in practice. The skillful handling of subjects such as health hints to dog owners, nutrition, geriatrics, and emergency procedures makes this film suitable for showing to a large variety of audiences that need to be informed on the role of the veterinarian in this branch of veterinary medicine.

This film has been cleared for television and is recommended for showing to lay and professional audiences.

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(WOMEN'S AUXILIARY—*continued from p. 22*)

they would like to hear a talk on modern veterinary science and also read or hear on the radio or TV about improved prevention and treatment of disease. Four out of five would like to hear on radio or TV information about the care of pets.

When radio directors and newspaper editors were questioned, four out of five said veterinarians were desired as guests on radio and TV but only 30 per cent found them to be sources of usable news. News of association activities was of slight value (5%) but 64 per cent were interested in animal disease outbreaks, unusual happenings, and activities that promote public health. A small town editor said: "The small town paper lives and dies on local news or the lack of it. If veterinarians can offer such, its good news to us." A radio director observes: "There are definite exceptions to the rule, but I find veterinarians among the most difficult to work with on radio. As individuals, they seem to have very little appreciation for public relations, which could be developed via radio."

There it is and here we are. Our task is cut out for us. We need to take a long serious look at ourselves. As we go about our work each day, are we each being a good public relations representative for the profession? Some of us have been taking the attitude that good public relations is something we buy when we pay association dues. The best paid public relations representative can not make the public think favorably of the profession if we fail to impress the man who pays for our services, as well as our neighbors. Each veterinarian and his wife, knowingly or unknowingly, are public relations representatives for the profession every day of their lives.

If 65 per cent of the radio directors and newspaper editors are interested in using material on veterinary topics, we are letting good public relations worth thousands of dollars slip through our fingers if we do not take advantage of this interest. Veterinarians need no previous radio experience to participate in a radio program if they use the seven-minute AVMA public information scripts. They are easy to read and phrased in language the average veterinarian would use. The topics discussed in the scripts are timely for the season of the year and the section of the country in which they are used.

Enterprising Minnesota auxiliary members contacted small town newspapers which resulted in many editors using the scripts on the farm page in an interview type of article. Farmers who may not have heard the program on the radio are likely to read the farm page of the local paper. The farmer or pet owner is made to feel that his local veterinarian is interested in his welfare, because the scripts not only tell "what to do until the doctor comes" but how to improve farm sanitation and prevent disease. Besides the radio scripts, the AVMA has material on almost any veterinary topic imaginable, for any type of program, which is available at your request.

Before the year is any older, let us each make a resolution to be "good public relations-minded"—to take advantage of the opportunities to better our professional standing in our own community.

s/MRS. L. J. MILLER

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Indiana Auxiliary.—The twelfth annual meeting of the Women's Auxiliary to the Indiana Veterinary Medical Association convened January 11-13 at the Severin Hotel, Indianapolis, with Mrs. George R. Burch, president, New Augusta, presiding. There was a record regis-

Officers elected at the twelfth annual meeting of the Women's Auxiliary to the Indiana Veterinary Medical Association are (standing, left to right)—Mrs. Raymond W. Worley, South Bend, second vice-president; Mrs. C. C. Donelson, Thorntown, first vice-president; Mrs. Frank O. Gossett, Greenfield, secretary; (seated, left to right)—Mrs. Howard Glass, treasurer; and Mrs. Lowell Hinchman, Glenwood, president.

tration of 142, with a growing state membership of 182. The guest of honor was Mrs. E. N. Moore, president of the Women's Auxiliary to the AVMA, who gave an address at the joint opening session and at the Auxiliary business meeting.

We were proud of Indiana's representation in the Women's Auxiliary to the AVMA. Mrs. Frank R. Booth, Elkhart, is secretary, Mrs. R. C. Klussendorf, Terre Haute, is a member of the nominating committee, and Mrs. J. J. Fishler, Elkhart, is a member of the radio committee.

The Auxiliary voted to contribute to the

(*Continued on p. 26*)

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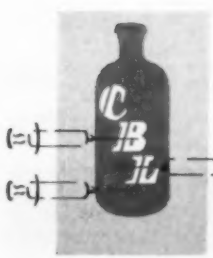

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(WOMEN'S AUXILIARY—*continued from p. 24*)

AVMA Research Fund, the Student Loan Fund, and the Memorial Fund.

The newly elected officers for the coming year are: Mrs. L. W. Hinchman, Glenwood, president; Mrs. C. C. Donelson, Thorntown, first vice-president; Mrs. R. W. Worley, South Bend, second vice-president; Mrs. Frank O. Gossett, Greenfield, secretary; and Mrs. Howard Glass, Indianapolis, treasurer.

The social events of the meeting were a smorgasbord, luncheon, and style show at the Highland Golf and Country Club, followed by a banquet and dancing.

s/MRS. FRANK O. GOSSETT, *Secretary.*

• • •

Minnesota Auxiliary.—The Women's Auxiliary to the Minnesota State Veterinary Medical Society held its annual meeting in St. Paul on Jan. 23-25, 1956. Mrs. W. L. Boyd presided at the business meeting, which followed a breakfast in the Hotel St. Paul. Mrs. D. B. Palmer, Wayzata, gave an interesting report on the AVMA Auxiliary meeting in Minneapolis. Mrs. C. E. Schrafel, Austin, reported on the progress of the radio public information programs of the AVMA—15 stations will broadcast the programs this year in Minnesota. The following contributions were approved: $75 to the library fund of the Minnesota School of Veterinary Medicine; $25 to the outstanding junior veterinary student; $15 to the Memorial Fund of the Women's Auxiliary to the AVMA in memory of Dr. B. A. Pomeroy; and $10 to the AVMA Research Fund.

Social activities included a tea, breakfast, coffee hour with the men, a banquet, and dance. At the tea, Mrs. F. E. Morse, Mankato, displayed her interesting collection of dolls and gave a talk on "International Dolls and International Friendships."

Officers elected for the coming year are: Mrs. E. H. Gloss, Gaylord, president; Mrs. E. P. Eder, Blue Earth, first vice-president; Mrs. G. E. Jacobi, Minneapolis, second vice-president; Mrs. A. P. Antroinen, Ortonville, secretary; and Mrs. R. W. Rieke, Fairmont, treasurer. Mrs. W. L. Boyd, St. Paul, is the retiring president.

s/MRS. A. P. ANTROINEN, *Secretary.*

STUDENT CHAPTER ACTIVITIES

California Chapter.—The first meeting of the University of California Student Chapter of the AVMA during the fall semester was an informal smoker held in September to introduce the incoming freshmen to the faculty and to other students. The attendance at this meeting was: seniors, 31; juniors, 29; sophomores, 35; and freshmen, 39—a total of 134.

On November 2, Dr. Larry Procter, Concord, Calif., spoke on methods of starting practice. The attendance was: seniors, 41; juniors, 28; sophomores, 32; and freshmen, 22—a total of 123.

At the December 7 meeting, V. Gray told of the history and current program of the California Dairy Breeders Association. The attendance of 134 included 40 seniors, 30 juniors, 28 sophomores, and 36 freshmen.

Plans for the midwinter conference for veterinarians to be held at the University were discussed at the Jan. 4, 1956, meeting. The 99 members present included 42 seniors, 21 juniors, 25 sophomores, and 11 freshmen. Dale Johnson took over the duties of president at this meeting.

s/JANET COPLAND-WENTZ, *Retiring Secretary.*

• • •

Ontario Chapter.—The Ontario Veterinary College Student Chapter of the AVMA met on Dec. 13, 1955. The guest speaker, Dr. T. L. Jones, principal of the Ontario Veterinary College, gave an excellent talk on his recent world tour as a member of a team of delegates investigating veterinary education and animal disease problems in the Far East. The tour was sponsored by the Food and Agriculture Organization of the United Nations.

After the meeting, coffee and doughnuts were served.

s/A. H. WOOD, *Past Secretary*

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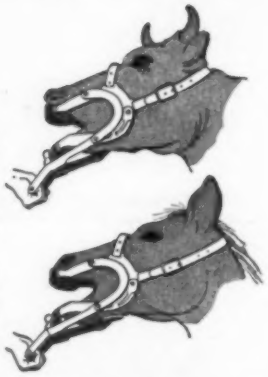

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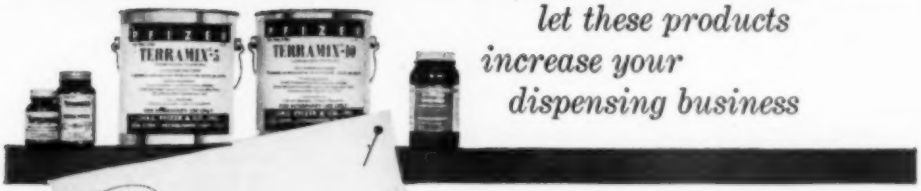

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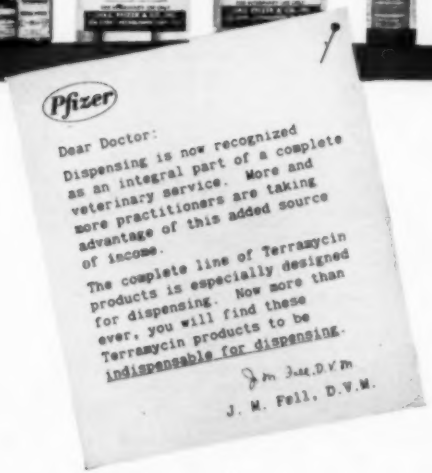
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COMING MEETINGS

Alabama Veterinary Medical Association. Annual meeting. Whitley Hotel, Montgomery, Ala., March 18-20, 1956. M. K. Heath, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala., secretary.

Arkansas Veterinary Medical Association. Annual meeting. Marion Hotel, Little Rock, March 27-29, 1956. W. M. Taegel, 906 Broadway, Little Rock, secretary.

Washington, State College of. Annual conference for veterinarians. College of Veterinary Medicine, State College of Washington, Pullman, Wash., April 2-4, 1956. John R. Gorham, conference chairman.

Animal Disease Research Workers in the Southern States. Annual meeting. Duncan Hall Auditorium, Alabama Polytechnic Institute, Auburn, April 5-6, 1956. Paul L. Piercy, University of Georgia, Athens, secretary.

Northern Illinois Veterinary Medical Association. Annual spring meeting. Hotel Faust, Rockford, Ill., April 18, 1956. J. M. Nelson, Sycamore Rd., Rt. 23, DeKalb, secretary.

Iowa, North Central Veterinary Medical Association. Annual meeting. Warden Hotel, Fort Dodge, April 19, 1956, at 10:00 a.m. H. Engelbrecht, Fort Dodge, secretary.

Eastern Iowa Veterinary Association, Inc. Annual all-day practitioners' clinic. Hawkeye Downs, Cedar Rapids, May 8, 1956. Forrest E. Brutsman, Traer, secretary.

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American Animal Hospital Association. Annual meeting. Hotel Fontainebleu, Miami Beach, Fla., May 23-26, 1956. W. H. Riser, 5335 Touhy Ave., Skokie, Ill., executive secretary.

California State Veterinary Medical Association. Annual meeting. Hotel Statler, Los Angeles, June 10-13, 1956. Charles S. Travers, 3004 16th St., San Francisco, executive secretary.

Maryland State Veterinary Medical Association. Annual summer meeting. George Washington Hotel, Ocean City, Md., June 28-29, 1956. John D. Gadd, Cockeysville, Md., secretary.

South Carolina Association of Veterinarians. Summer meeting. Clemson House, Clemson College, S. Car., June 28-30, 1956. Worth Lanier, York, S. Car., secretary.

Mississippi State Veterinary Association. Annual meeting. Buena Vista Hotel, Biloxi, July 15-17, 1956. Harvey F. McCrory, P.O. Box 536, State College, secretary.

Iowa State College. Annual conference for veterinarians. Memorial Union, Iowa State College, Ames, July 17-18, 1956. John B. Herrick, Iowa State College, Ames, co-chairman of conference.

Alabama Polytechnic Institute. Annual conference for veterinarians. School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala., July 22-25, 1956. R. S. Sugg, dean.

Pennsylvania State Veterinary Medical Association. Annual meeting. Bedford Springs Hotel, Bedford, Pa., Sept. 12-14, 1956. Raymond C. Snyder, N.W. Corner Walnut St., and Copley Rd., Upper Darby, Pa., secretary.

Colorado Veterinary Medical Association. Annual meeting. Estes Park, Sept. 7-9, 1956. G. H. Gilbert, 5500 Wadsworth Blvd. Arvada, Colo., secretary.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Montrose, Cedar Rapids, Oct. 4-5, 1956. Forrest E. Brutsman, Traer, secretary.

American Veterinary Medical Association. Annual meeting. Municipal Auditorium, San Antonio, Texas, Oct. 15-18, 1956. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

U. S. Livestock Sanitary Association. Annual meeting. Morrison Hotel, Chicago, Ill., Nov. 28-30, 1956. R. A. Hendershott, 33 Oak Lane, Trenton 8, N. J., secretary.

Foreign Meetings

World Congress on Fertility and Sterility. Naples, Italy, May 18-26, 1956. Professor T. Bonadonna, Via Monte Ortigara, 35, Milan, Italy, in charge of Veterinary Section.

Tenth International Congress of Entomology. McGill University and University of Montreal, Montreal, Canada, Aug. 17-25, 1956. J. A. Downes, Division of Entomology, Science Service Bldg., Ottawa, Ont., Canada, secretary.

International Association of Hydatidology. Sixth Congress. Athens, Greece, Sept. 14-18, 1956. Prof. B. Kourias, 1 MacKenzie King St., Athens, Greece, general secretary.

(*Continued on p. 30*)

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References: 1. Brown, A. L., et al.: Vet. Med. 50: 167, April 1955. 2. Agricultural Research Service, Losses in Agriculture, June 1954, Table 20, p. 129.

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(COMING MEETINGS—*continued from p. 28*)

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. G. J. Phelps, Jr., Montgomery, Ala., secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. W. R. Laster, Jr., 213 N. 15th St., Birmingham, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Pima County Veterinary Medical Association, the third Wednesday of each month in Tucson. E. T. Anderson, 8420 Tanque Verde Rd., Tucson, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Wilfred Pimentel, 3455 S. Elm Ave., Fresno, Calif., secretary.

East Bay Veterinary Medical Association, bimonthly, the fourth Wednesday. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. B. C. Watson, 825 14th St., Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Thursday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at Eaton's Restaurant in Studio City, Calif. R. A. Button, 5954 Van Nuys Blvd., Van Nuys, Calif., secretary.

Southern California Veterinary Medical Association, the third Wednesday of each month. Howard C. Taylor, 2811 West Olive St., Burbank, Calif., secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of

(*Continued on p. 32*)

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Calf Scours	Infectious Canine Hepatitis	Pyelonephritis
Calf Diphtheria	Lamb Diarrhea	Septic Sore Throat
Canine Distemper (Secondary Infection Of)	Laryngitis	Septicemia
	Leptospirosis	Strangles
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- **causes fewer gastrointestinal side effects.**
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*Newcomer, V. D., Wright, E. T., and Sternberg, T. H.: Antibiotics Annual 1954-55, New York, Medical Encyclopedia, Inc., 1955, p. 686.

administration and dosage: **Administration is by the oral route. Dosage (daily, in divided doses):**
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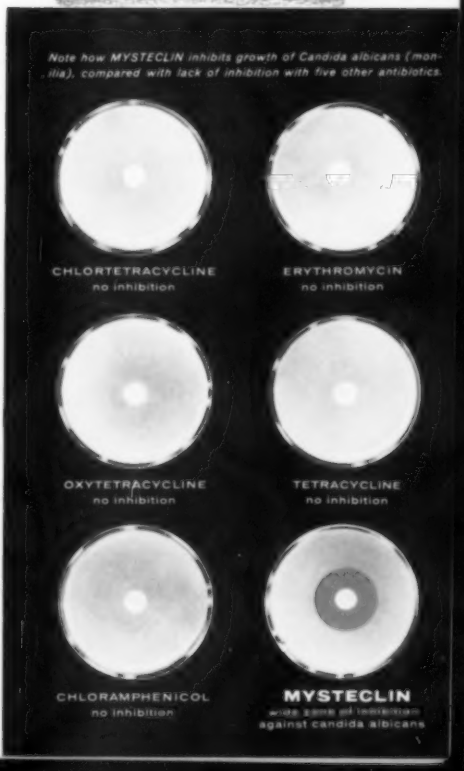

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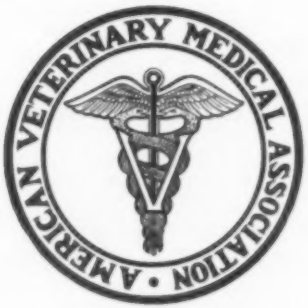

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Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Jacksonville Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. George F. Yopp, 4644 Main St., Jacksonville, Fla., Secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. Ross E. Evans, 5215 S. Dixie Highway, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. R. P. Link, College of Veterinary Medicine, University of Illinois, Urbana, Ill., secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Charles J. York, P. O. Box 1656, Indianapolis 6, Ind., secretary.

Michiana Veterinary Medical Association, the second Thursday of each month, at the Hotel LaSalle, South Bend, Ind. L. D. Ramsay, 719 E. Jefferson Ave., La Porte, Ind., secretary.

Tenth District Veterinary Medical Association the third Thursday of each month. W. E. Sharp, Union City, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. D. A. Buchanan, Grundy Center, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KENTUCKY—Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. Dr. W. E. Bewley, P. O. Box "H", Crestwood, Ky., secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

(*Continued on p. 36*)

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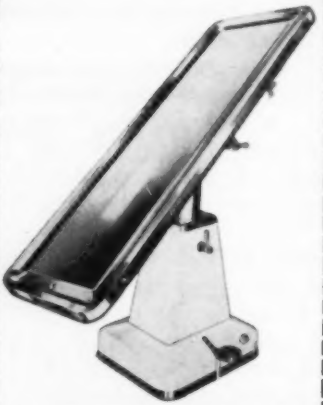

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to help you fight stubborn bovine mastitis

Statistics show that organisms other than the more common streptococcus and staphylococcus are causing troublesome mastitis in today's herds, in ever-increasing numbers of cases. *Pseudomonas aeruginosa, Corynebacterium pyogenes, E. coli* and the *Proteus* groups are some of these organisms, which will not succumb to the more frequently-used antibiotics. We are also finding that staph and strep are becoming more resistant to conventional treatment.

NEO-600 contains more than 600 mg. of neomycin sulfate (equivalent to 350 mg. of neomycin base), which is the recommended dosage for the treatment of *Ps. aeruginosa, E coli, Str agalactiae, Str. dysagalactiae, Str. uberis* and *Micrococcus pyogenes* var. *aureous*.[1]

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NEO-600 is a new and effective weapon in your fight against mastitis.

References:

[1] Drury, A.R.: Vet. Med. 47: 407-411, 1952.

[2] Kitchen, H., and Waksman, S., J.A.V.M.A., Vol. 127, No. 942: 261-274, Sept., 1955.

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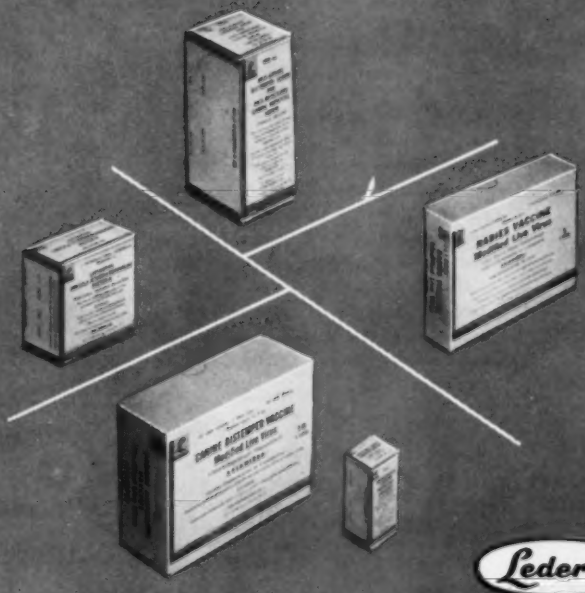

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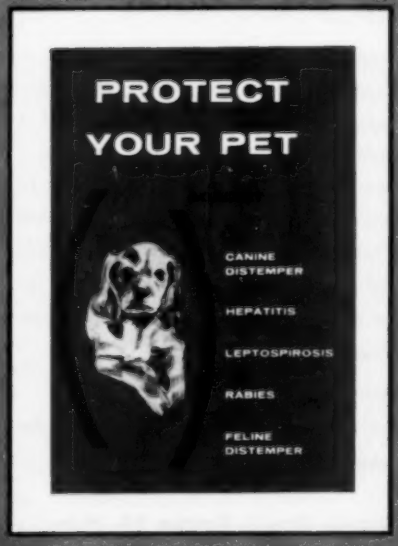

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Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck, N. J. Edward Baker, 568 Grand Ave., Englewood, N. J., secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel in Greensboro, N. Car. J. W. Peace, 507 E. Homestead Ave., High Point, N. Car., secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. Wm. Allen Potts, 401 W. James St., Mount Olive, secretary.

Piedmont Veterinary Medical Association, the last Friday of each month at 7:00 p.m. in Mull's Motel in Hickory, N. Car. W. W. Dickson, Box 1071, Gastonia, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month except July and August. James M. Brown, 2818 W. Britton Rd., Oklahoma City, secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Merle S. Watts, 5302 E. 11th St., Tulsa, Okla., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fair-

(*Continued on p. 37*)

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(COMING MEETINGS—*continued from p. 36*)

forest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R. Des Rosiers, 5508 2nd Ave., N. W., Seattle 7, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. Jo Walker, Agriculture Experiment Station, Puyallup, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W., Huntington, W. Va., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

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(*Continued on p. 40*)

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(*Continued on p. 42*)

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No parts of dilator will remain in teat canal.

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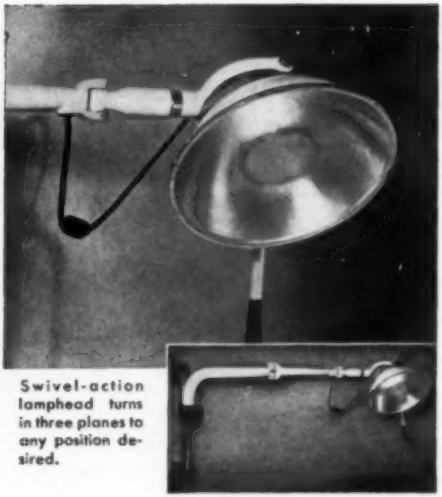

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(CLASSIFIED ADS—*continued from p. 40*)

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(*Continued on p. 44*)

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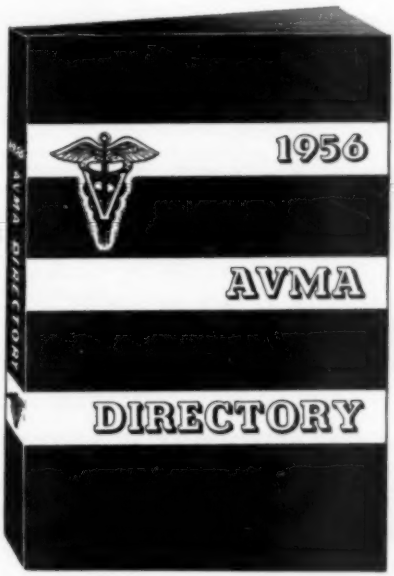

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(CLASSIFIED ADS—*continued from p. 42*)

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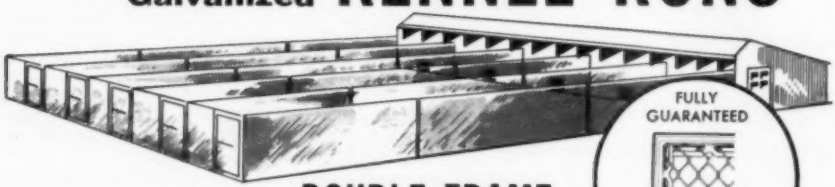

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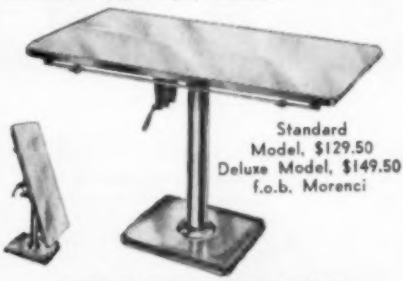
Features precision-made hydraulic unit guaranteed for life of the table. Finger-touch control raises, lowers and locks table in seconds. Large 24" x 60" top. Movable on concealed casters.

STANDARD MODEL
$99.50

DELUXE MODEL
$129.50

EXAMINATION TABLE

Reinforced stainless steel top in 22" x 48" size. Six tie clamps. Patented "corner compensators" automatically level table on any surface. Designed for examination and pre-surgery treatment.



Standard Model, $129.50
Deluxe Model, $149.50
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STAINLESS STEEL OPERATING TABLE

Chrome plated single pedestal and base combined with reinforced stainless steel top. Positive locking adjustment in twelve positions. Super size 24" x 60" top.

ALL MODELS AVAILABLE FROM LEADING SUPPLIERS

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for highly effective treatment of bacterial infections in large animals, small animals, and poultry.

SULBEN'S wide scope of indications for use includes many common diseases of farm animals, poultry and pets wherein sulfa-sensitive organisms are the etiological agents; particularly enteric conditions and those due to Streptococcus, Staphylococcus, and Pasteurella organisms respond to Sulben. It is economical and dependable, a single dose often being sufficient.

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SULBEN is available in six forms:

Sulben Solution (*for oral use*)
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HAVER-GLOVER *Laboratories*
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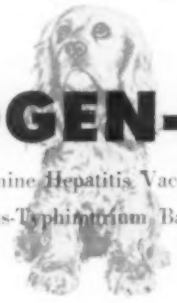
ALOGEN

Canine Distemper Vaccine in Bronchisepticus-Streptococcus-Typhimurium Bacterin.

Introducing two new members of the
***Lockhart** canine vaccine family*



ALOGEN-H

Canine Distemper Vaccine and Infectious Canine Hepatitis Vaccine in Bronchisepticus-Streptococcus-Typhimurium Bacterin.

ALOGEN and ALOGEN-H are brought to you in 5 cc. disposable cartridges. **You may use these with your regular syringe just as you would any other vial, or with the new *AUTOJET* syringe.**

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Jen-Sal

modified live virus **hog cholera vaccine**

SV-2

SV-2

gives **maximum long-term protection**

with **minimum reaction risks**

SV-2

* **Simultaneous Virus #2**

Administered with a minimal dose of serum, Jen-Sal SV-2 provides immediate, durable immunity without postvaccination virus flare-ups or live virus premise contamination. Supplied in 2, 5, 10, 25 or 50 dose vials.

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